The Newsletter of the Australian and New Zealand Society of Paediatric Dentistry

Synopses

November 2019, ISSUE 65

Non-restorative cavity treatment: is it the only way?

Dr Amanda Margareta Leske
Doctor of Clinical Dentistry (Paediatric Dentistry) Candidate
The University of Melbourne

Abstract

Non-restorative cavity treatment is one of the many options for managing dental caries in the primary dentition. With better understanding of the caries process, societal shifts in parenting styles and increasing costs associated with conventional dental care, non-restorative treatment options are likely to appeal to policymakers, clinicians, children and parents alike. However, it is important that dental practitioners perform a judicious assessment of the available evidence-base, prior to recommending management options for carious teeth. This review critically discusses a recent opinion piece and highlights potential benefits and risks of prescribing non-restorative caries management.

Introduction

Dental caries is one of the world's most prevalent chronic diseases. An estimated 486 million children suffer from dental caries in their primary dentition, with the majority being untreated cavities[1]. In Australia, approximately half of all children will experience caries by school-age[2]. Despite widespread knowledge and resources for prevention and management of dental caries, data from global epidemiological studies demonstrates an increase in the disease prevalence in children[3-8]. Recently, especially in the paediatric dental literature, there has been a trend towards non-restorative cavity treatment (NRCT), or biological management of dental caries. With increasing populations, inflated cost of dental care, stressed healthcare systems, changing parenting styles and the shift from paternalistic surgical dental care to one that considers patient- and family-based outcomes, NRCT might represent a significant advantage over other caries management options. However, the evidence-base for such practice is still in its infancy, meaning that expert opinion is currently relied upon to guide decision and policymaking in this area. This review will provide a critical appraisal of a recent opinion article published in Dental Update in 2019, entitled "Non-restorative cavity treatment: should this be the treatment of choice? Reflections of a teacher in paediatric dentistry" 9 by René Jm Gruythuysen, who is a PhD qualified retired dentist and post-academic teacher in paediatric dentistry from Rotterdam, The Netherlands. The article provides a strong persuasive stance supporting NRCT; advising that, because caries is a biological disease, biological treatment options should be offered in the first instance. Furthermore, conventional restorative treatment should be considered a symptomatic, less child-friendly option that merely masks the caries activity.

Relevance, timeliness and novelty

This opinion piece is relevant and timely, congruent with the relative growth in the recent paediatric dental literature regarding non-restorative options for the management of carious lesions. It is

continued on page 3...

THIS ISSUE

by Dr Sue Cartwright

BDS, Dip Clin Dent, M Ed

Everyone deserves a future they **can smile about.**

Sadly, all good things must come to an end. It is with regret that we are announcing the end of Colgate support for the production of Synopses.

We have looked after this journal for many years providing graphic design, printing and distribution services. It has been a pleasure to be able to assist with the dissemination of dental paediatric research and news. The articles are always interesting and of very high quality. We are honoured to partner with such a strong association that provides great support for its members and is invested in keeping people up to date with current trends and learning.

Synopses has been produced by Colgate for ANZSPD from the early 1990s. A newsletter like this is so important to keep the dental paediatric community informed and we wish you all the best with the continuation of this important initiative.

Although this will be the final newsletter to be produced by Colgate, we hope to continue our strong relationship with ANZSPD through other means, such as conference sponsorship and prizes for academic competitions.

Thank you for allowing us the privilege of supporting this newletter for so many years. It has been great!

The Newsletter of the Australian and New Zealand Society of Pediatric Dentistry

Synopses

December 1991, Issue 2

Fanconi's anaemia with gingival hyperplasia

A case report

Coming events

Inside

President's Report	page 2
Branch News	page 3
The Robin Sequence	page 6
Interchange	page 8
Meeting Reports	page 12

Front cover of early edition.

Colgate Oral Care Consultants are here to assist you with the products you need in your surgeries

NSW
Louise Clarke 0419 993 700
Louise McAllister 0408 409 545
Rochelle Spargo 0410 488 581

QLD
Narelle Bird 0417 642 665
Marcelle Johnson 0457 772 997

VIC
Catherine Bensa 0417 598 170
Sabrina Moey 0427 440 232
Kerryn Pope 0458 280 739

SA & NT
Leanne Nelson 0400 387 249

WA
Kim Savory 0400 505 223
Kiri Stowell 0408 997 366

NEW ZEALAND
Michelle Jelley 021 621 315
Debra Morrissey 021 593 986

Colgate National Sales Managers

Australia Nolene Devery 0419 998 515
New Zealand Lisa Fraser 021 593 985

Orders for Colgate products are placed through Henry Schein Halas

Phone: 1300 65882 Fax: 1300 658810

...continued From page 1

well known that the implementation of minimally invasive dentistry (MID) into dental practice has been met with some resistance, and the reasons for this have yet to be fully elucidated[10]. Considering the initial and ongoing difficulties in acceptance and implementation of other MID techniques in primary teeth, such as delaying restorative intervention[11], selective carious tissue removal[12] and the Hall technique[13], NRCT will likely be met with similar opposition from a large part of the established paediatric dental community.

The author raises some novel points, in particular the use of the psychological measure of locus of control, of both the patient/parent and dental provider, as an influencing factor for the successful implementation of NRCT into dental practice[14]. This dimension of locus of control refers to the extent to which people believe they can influence the events in their lives and is often simplified as internal or external. The locus of control of the practitioner might influence how well they accept integration of the technique into their practice and that of the patient/parent will likely influence their ability to successfully manage the carious lesions with at-home oral hygiene practices[15]. Further psychological dimensions of clinician and patient behaviour, such as self-efficacy, should be researched in implementation studies, as it is likely that these will have a significant influence on the success of implementation of MID treatment options into dental practice.

There are no systematic reviews that adequately appraise the evidence of NRCT due to lack of studies and heterogeneity in techniques and outcomes. The most recent systematic review and network meta-analysis on non-restorative treatments for caries identified the heterogeneity of studies and reported that the lack of a common comparator across non-restorative interventions prevented the creation of a network[16]. Therefore, expert opinion, as provided by Gruythuysen[9], is currently required to synthesise what evidence is available and make sense of it to dental practitioners and policymakers.

While the article provides a persuasive argument supporting implementation of NRCT into paediatric dental practice, it fails to identify any clinical situations where this procedure might not be suitable and the appropriate alternatives. This is likely to be detrimental when teaching students and general dental practitioners to critically consider all the restorative and non-restorative options available to paediatric patients.

Aetiology and consequences of dental caries

The current definition of dental caries as a mostly preventable, behaviourally driven, biofilm-mediated, sugar-driven, multifactorial, dynamic disease that results in the imbalance of demineralization and remineralization of dental hard tissues[17] highlights that its appropriate management involves not only input from dental professionals but also from well-informed parents, non-dental health professionals, community health workers and evidence-based health policy. There are numerous potentially devastating consequences of dental caries including pain, bacteraemia, eating and speech problems, learning difficulties, malocclusion, growth disturbances, activity restriction and dysfunctional family and peer relationships1[8-21]. Treatment of caries places an immense burden on the healthcare system and is a common cause of potentially preventable hospitalisations[22]. In Australia, oral conditions are the second most expensive disease group to treat after cardiovascular disease, with $10.2 billion dollars being spent on dental treatment in 2016-2017[23]. Children aged five to nine years have the highest rates of both potentially preventable hospital separations and general anaesthetic hospital separations for treatment of oral conditions, resulting in approximately 30,000 Australian children per year requiring hospital treatment for dental conditions, often related to dental anxiety or special needs[2]. As the author correctly highlights, NRCT might provide an appropriate solution to many of the challenges associated with providing dental treatment to children, however, it should not be considered the only option and in many cases, it is likely to present risks and adverse effects to the child and their quality of life. Children should not be denied restorative treatment simply because they are difficult to treat, and resources need to be directed into strategies to improve training, funding and facilities to provide efficacious dental treatment to all children.

Efficacy of conventional restorative treatment in primary teeth

It has long been known that restoring carious lesions does little to change caries risk [24]. The author takes a very firm stance against restorative treatment in children, stating that "restorative treatment of caries lesions in a child with neglected teeth masks the lack of oral health care"[9]. However, the article fails to consider the proven success of restorative treatment in primary teeth, when executed with skill and according to the evidence-base[25]. The article displays a clinical photograph of a tooth with a failed tooth-coloured restoration on an extracted tooth, explaining that "perfect restoration could not prevent the development of a new carious lesion after two years" 9. However, the literature at present shows equivocal failures of conventional direct restorations in primary teeth and NRCT[26]. It could be argued that the placement of the restoration delayed pulpal infection and allowed development of the child to an age where more complex treatment such as extraction could be tolerated with minimal consequences. Additionally, it is not appropriate to compare NRCT to a procedure that has a known high risk of failure in primary teeth of high caries risk patients, such as direct resin composite restorations[27]. All this reveals is that NRCT is superior to poor restorative and preventive dentistry, which is not surprising or relevant.

Efficacy of non-restorative cavity treatment

The evidence supporting NRCT for the management of carious lesions in primary teeth can, as yet, be considered weak, highlighted by the limited number of clinical trials cited by the author [15, 26,28,32]. The author implies that the majority of failures associated with NRCT is likely to be incorrect slicing technique, and that most studies do not provide a clear protocol regarding the clinical procedure. However, the literature has yet to divulge all the potential reasons for failure of NRCT, including both patient, parent, tooth and operator factors, and these will need to be considered by a dental practitioner on a case-by-case basis.

Before NRCT can be recommended as a treatment of choice, it will require a large number of patients and treatments to be

analysed, ideally in a systematic review and meta-analysis, with a focus not only on the efficacy in comparison to conventional restorative treatment but also on the potential for adverse effects. Phase III trials generally recommend between 1000-5000 patients to be analysed in order to determine potential adverse effects and perform a risk-benefit analysis of the procedure. The extant literature contains, at most, only a few hundred cases, although this is increasing. The problem, as the author correctly identifies, is that patients in studies examining NRCT will be difficult to randomise, as it will likely run into ethical dilemmas regarding patient and parent preferences and controlling for the numerous variables that might influence progression of a carious lesion. A core set of outcomes for caries studies should help in this regard, to make meta-analysis of studies more readily possible[33].

Adverse effects of NRCT

The adverse effects of NRCT are not adequately addressed, with criticisms of the technique being addressed without supporting references, such as the recommendation to apply fluoride to eliminate sensitivity after NRCT. It should not be forgotten that untreated dental caries and cavitated lesions can have significant consequences on aesthetics, comfort, function, occlusion, development of permanent teeth and general health and wellbeing. There have been reports of child deaths from spreading dental abscesses[34]. These factors are not addressed in the article and has the potential to mislead dental practitioners into ignoring the risks of cavitated carious lesions encroaching on the pulp, particularly in immunocompromised and neglected children. The importance of a thorough medical, dental and social history should not be underestimated when considering NRCT an option for a child, particularly if they do not have motivated caregivers that can assist in the remineralisation and monitoring of the lesions.

Non-restorative cavity treatment is not suitable for all children

The author identifies the indications for non-restorative cavity treatment; however, fails to clearly outline contra-indications for the technique. This could inadvertently stimulate less experienced clinicians to implement this technique for all child patients. Children who are immunocompromised or with significant cardiac conditions, for example, are unlikely to be suitable candidates for this technique, due to the risk of bacterial penetration into the bloodstream and surrounding tissues[35]. Due to the lack of widespread dissemination of the potential success of this technique outside the paediatric dentistry community, it is unclear whether medical practitioners or other health professionals are aware of the technique and their opinions on the medical risks of such procedures are currently unexplored. Before implementing NRCT on a medically compromised child, the opinion of the treating medical practitioner must be sought. This further stresses the need for parents and patients to be informed in detail of the treatment plan and aims, to prevent confusion and re-referral of patients back to dental practitioners from medical practitioners who are unaware that cavitated lesions are actually in active treatment.

The article suggests that the oral health and well-being of children can best be served by NRCT. However, this might not be best for every child or every family. There are likely to be significant failure rates in children without the availability of motivated parents/caregivers to assist with oral hygiene and higher dissatisfaction rates for children and families who have high aesthetic expectations, who experience bullying related to discoloured or fractured teeth or who experience functional difficulties.

Patient preferences and abilities

Dental researchers are realising the importance of patient-based outcomes in intervention studies and are more commonly including these in studies on management of carious lesions[36]. Interestingly, NRCT has often shown no difference compared to restorative management options in terms of patient and clinician acceptability [29]. This article fails to address the fact that patient and family preferences must be taken into account when prescribing restorative and non-restorative dental treatments, as evidence-based dentistry is known to encompass not only the best available evidence but also what is best for the patient in that particular circumstance[37]. It would not be ethical to only offer NRCT when other treatment options are available and are known to provide suitable outcomes, particularly in regard to aesthetics and function. Patients and parents who can cope and regularly access conventional dental treatment in the dental chair might be confused and dissatisfied by the abrupt change to NRCT. Culture, values, self-efficacy and abilities of individual patients are important to assess as these can also have a significant impact on the success of NRCT[14]. Cross-cultural differences also need to be explored, as there is evidence of a high acceptance rate of non-restorative options such as silver diammine fluoride (SDF) in certain cultures which might not translate across other populations[38].

Operator skill and experience

It is not uncommon to encounter dental practitioners who advocate not restoring primary teeth due to their exfoliative nature, combined with their uncertainty in treating children due to potential anxiety and lack of cooperation. However, there is a big difference between actively and passively leaving a carious primary tooth to exfoliate and, subsequently, a fine line between supervised remineralisation and supervised neglect. Before NRCT can be recommended as the treatment of choice, clear, practical and visual guidelines will need to be developed and disseminated to general dental practitioners, ideally with hands-on training courses, in both the practical side of performing the slicing and the behavioural guidance techniques needed to make the treatment a success. Appropriate evidence-base and resultant guidelines will also need to be developed to advise when it might be appropriate to combine NRCT with adjunctive techniques such as fluoride varnish, SDF or atraumatic restorative technique.

Behaviour change

Behavioural change interventions can be defined as coordinated sets of activities designed to change specified behaviour patterns[39]. Behaviour change of high caries risk child patients and their families is considered a complex intervention. The author appears to simplify the behaviour change technique of motivational interviewing, which

has been shown to take time, extensive training and practice to implement and there is, as yet, little evidence to support its efficacy in combination with NRCT or, in fact, any restorative or non-restorative caries management regime when performed by dental practitioners in clinical dental practice[40]. Two recent, large scale, good quality trials have shown no effect of motivational interviewing when compared to traditional oral health education in preventing the development of carious lesions[41,42]. It might be overly optimistic for the author to assume that behaviours that led to the development of cavitated carious lesions will suddenly be modified to the extent that will lead to arrest of the carious process.

It takes a village

For successful implementation of NRCT it will be important to inform and include many involved in the child's life, including all parents and carers, medical practitioners, maternal and child health nurses, daycare/school staff and the whole dental team, including support staff. Without the involvement and support of these key stakeholders, the success of NRCT is likely to be affected. Multiple opportunities to reinforce healthy dietary habits, oral hygiene and prevention of caries should not be missed and all participants need to be aware of the procedure, its aims and potential benefits, as well as looking out for warning signs that it might be failing. Further efforts will be needed to improve the oral health promotion skills of non-dental health professionals, as it is known that these providers often are unwilling to take responsibility for the oral health care of young children due to lack of confidence and training[43]. More research will need to be done into the level of support that is required for families to be effective in fulfilling their role as primary preventers of dental caries, particularly in families of socio-economic disadvantage, so that NRCT does not further exacerbate social inequality in dental health.

Cost and time effectiveness

The cost and time effectiveness of NRCT has not yet been proven superior to other caries management options. The time taken to provide efficacious oral hygiene instruction and repeatedly review the lesions might preclude cost-effectiveness. One German randomised controlled trial discovered that the Hall technique was more cost-effective than NRCT for managing cavitated carious lesions in primary molars, yielding better dental health outcomes at lower costs, and reported that NRCT cost-effectiveness was similar to conventional restorations [31]. Although personal experience might advise that NRCT avoids dental treatment under general anaesthesia, this has yet to be determined by empirical research. Additionally, when carefully planned and executed by highly trained paediatric dental specialists, there is evidence that dental treatment performed under general anaesthesia has high success rates and low repeat rates, although unfortunately this is often not the case in real world clinical practice [44]. It might be considered that it is often not the restorative treatment that fails, but the behaviour change techniques and efficacy, which can potentially fail in both restorative and non-restorative treatment regimes.

Another facet not explored by the article is that current fee schedules and design of dental practices preclude the financial viability and motivation to provide NRCT. For NRCT to be successfully implemented into routine dental practice, a dramatic restructure of the dental system would be required. Fee schedules would need to recognise the time-consuming and challenging task of eliciting patient behaviour change, which should be considered as important as any surgical treatment provided. This might require the division of dental practitioners into 'biological' dentists, who provide NRCT, with referral to 'surgical' dentists who provide invasive restorative/operative management when required, similar to the medical model currently in the Australian healthcare system. This would ensure optimal skills and practice in each of the sub-specialities and provide patients with a better understanding of the management of their disease.

Conclusion

The integration of MID into dental practice requires a significant shift in the delivery of oral healthcare from a largely surgical model to one that focuses on the biological management of the disease and recognises the essential role patients and families have in maintaining their own oral health. It is likely that NRCT will have a place in the future of dental treatment, but more research is required into the aspects of the clinical and behavioural techniques that make it successful and potential short- and long-term adverse effects. Once this evidence is available, there is likely to be a long implementation period where dental practitioners will need to be convinced and trained to perform the technique correctly and where patients, parents and health professionals will need to be fully informed of the potential benefits and their relation to all the other caries management options available.

Why is this paper important to paediatric dentists?

• This paper critically summarises a recent opinion piece and discusses the potential benefits and risks of non-restorative cavity treatment

• This paper highlights the need for more research into the clinical and behavioural factors that might make non-restorative cavity treatment most successful

REFERENCES

1. Global Burden of Disease 2016 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet (London, England) 2017;390:1211-1259.
2. Chrisopoulos S, Harford J, Ellershaw A. Oral health and dental care in Australia: key facts and figures 2015. Cat. no. DEN 229. Canberra: AIHW, 2016.
3. Dye BA, Arevalo O, Vargas CM. Trends in paediatric dental caries by poverty status in the United States, 1988-1994 and 1999-2004. Int J Paediatr Dent 2010;20:132-143.
4. Bagramian RA, Garcia-Godoy F, Volpe AR. The global increase in dental caries. A pending public health crisis. Am J Dent 2009;22:3-8.
5. Villalobos-Rodelo JJ, Medina-Solis CE, Molina-Frechero N, Vallejos-Sanchez AA, Pontigo-Loyola AP, Espinoza-Beltran JL. Dental caries in schoolchildren aged 6-12 years in Navolato, Sinaloa, Mexico: experience, prevalence, severity and treatment needs. Biomedica : revista del Instituto Nacional de Salud 2006;26:224-233.
6. Tsai AI, Chen CY, Li LA, Hsiang CL, Hsu KH. Risk indicators for early childhood caries in Taiwan. Community Dent Oral Epidemiol 2006;34:437-445.
7. Haugejorden O, Magne Birkeland J. Ecological time-trend analysis of caries experience at 12 years of age and caries incidence from age 12 to 18 years: Norway 1985-2004. Acta odontologica Scandinavica 2006;64:368-375.
8. Armfield JM, Spencer AJ, Brennan DS. Dental health of Australia's teenagers and pre-teen

children: The Child Dental Health Survey, Australia 2003–04. Dental statistics and research series. Canberra: AIHW, 2009.
9. Gruythuysen RJ. Non-restorative cavity treatment: should this be the treatment of choice? Reflections of a teacher in paediatric dentistry. Dental Update 2019;46:220-228.
10. Banerjee A. 'Minimum intervention' - MI inspiring future oral healthcare? Br Dent J 2017;223:133-135.
11. Laske M, Opdam NJM, Bronkhorst EM, et al. Minimally Invasive Intervention for Primary Caries Lesions: Are Dentists Implementing This Concept? Caries Res 2019;53:204-216.
12. Innes N, Robertson M, Schwendicke F. Caries Excavation: Evidence Gaps. Monographs in oral science 2018;27:167-171.
13. Innes NP, Evans DJ, Bonifacio CC, et al. The Hall Technique 10 years on: Questions and answers. Br Dent J 2017;222:478-483.
14. Duijster D, de Jong-Lenters M, Verrips E, van Loveren C. Establishing oral health promoting behaviours in children - parents' views on barriers, facilitators and professional support: a qualitative study. BMC oral health 2015;15:157-169.
15. Hansen NV, Nyvad B. Non-operative control of cavitated approximal caries lesions in primary molars: a prospective evaluation of cases. J Oral Rehabil 2017;44:537-544.
16. Urquhart O, Tampi MP, Pilcher L, et al. Nonrestorative Treatments for Caries: Systematic Review and Network Meta-analysis. J Dent Res 2019;98:14-26.
17. Pitts N, Baez, R., Diaz-Guallory, C. Early Childhood Caries: IAPD Bangkok Declaration. Pediatr Dent 2019;41:176-178.
18. Low W, Tan S, Schwartz S. The effect of severe caries on the quality of life in young children. Pediatr Dent 1999;21:325-326.
19. Casamassimo PS, Thikkurissy S, Edelstein BL, Maiorini E. Caries: Beyond the dmft: The human and economic cost of early childhood caries. Journal of the American Dental Association 2009;140:650-657.
20. Blumenshine SL, Vann WF, Gizlice Z, Lee JY. Children's school performance: impact of general and oral health. Journal of public health dentistry 2008;68:82-87.
21. Law E, Fisher E, Eccleston C, Palermo TM. Psychological interventions for parents of children and adolescents with chronic illness. The Cochrane database of systematic reviews 2019;3:Cd009660.
22. Australian Institute of Health and Welfare. Healthy Communities: Potentially preventable hospitalisations in 2013–14. https://www.aihw.gov.au/reports-data/indicators/healthy-community-indicators: Australian Institute of Health and Welfare, 2015.
23. Australian Institute of Health and Welfare. Oral health and dental care in Australia. Cat. no: DEN 231. https://www.aihw.gov.au/reports/den/231/oral-health-and-dental-care-in-australia/contents/summary: Australian Institute of Health and Welfare, 2019.
24. Kidd E. Should deciduous teeth be restored? Reflections of a cariologist. Dent Update 2012;39:159-162, 165-156.
25. Seale NS, Randall R. The use of stainless steel crowns: a systematic literature review. Pediatr Dent 2015;37:145-160.
26. Mijan M, de Amorim RG, Leal SC, et al. The 3.5-year survival rates of primary molars treated according to three treatment protocols: a controlled clinical trial. Clinical oral investigations 2014;18:1061-1069.
27. Chisini LA, Collares K, Cademartori MG, et al. Restorations in primary teeth: a systematic review on survival and reasons for failures. Int J Paediatr Dent 2018;28:123-139.
28. Leal SC, Bronkhorst EM, Fan M, Frencken JE. Effect of different protocols for treating cavities in primary molars on the quality of life of children in Brazil – 1 year follow-up. International Dental Journal 2013;63:329-335.
29. Santamaria RM, Innes NP, Machiulskiene V, Evans DJ, Alkilzy M, Splieth CH. Acceptability of different caries management methods for primary molars in a RCT. Int J Paediatr Dent 2015;25:9-17.
30. Santamaria RM, Innes NPT, Machiulskiene V, Schmoeckel J, Alkilzy M, Splieth CH. Alternative Caries Management Options for Primary Molars: 2.5-Year Outcomes of a Randomised Clinical Trial. Caries Res 2018;51:605-614.
31. Schwendicke F, Krois J, Splieth CH, et al. Cost-effectiveness of managing cavitated primary molar caries lesions: A randomized trial in Germany. J Dent 2018;78:40-45.
32. Vermaire JH, van Loveren C, Brouwer WB, Krol M. Value for money: economic evaluation of two different caries prevention programmes compared with standard care in a randomized controlled trial. Caries Res 2014;48:244-253.
33. Schwendicke F, Lamont T, Innes N. Outcomes in Trials for Management of Caries Lesions (OuTMaC): protocol. Trials 2015;16:397-397.
34. Otto M. How can a child die of toothache in the US? https://www.theguardian.com/inequality/2017/jun/13/healthcare-gap-how-can-a-child-die-of-toothache-in-the-us, 2012.
35. Foster H, Fitzgerald J. Dental disease in children with chronic illness. Archives of Disease in Childhood 2005;90:703-708.
36. Hayes C. The use of patient based outcome measures in clinical decision making. Community dental health 1998;15:19-21.
37. Sackett DL. Evidence- based medicine. Seminars in Perinatology 1997;21:3-5.
38. Chu CH, Lo ECM, Lin HC. Effectiveness of Silver Diamine Fluoride and Sodium Fluoride Varnish in Arresting Dentin Caries in Chinese Pre-school Children. Journal of Dental Research 2002;81:767-770.
39. Michie S, van Stralen MM, West R. The behaviour change wheel: A new method for characterising and designing behaviour change interventions. Implementation Science 2011;6:42.
40. Randall CL. On Motivational Interviewing for Oral Health Promotion: State of the Field and Future Directions. JDR Clinical & Translational Research 2018;3:376-377.
41. Henshaw MM, Borrelli B, Gregorich SE, et al. Randomized Trial of Motivational Interviewing to Prevent Early Childhood Caries in Public Housing. JDR Clinical & Translational Research 2018;3:353-365.
42. Batliner TS, Tiwari T, Henderson WG, et al. Randomized Trial of Motivational Interviewing to Prevent Early Childhood Caries in American Indian Children. JDR Clinical & Translational Research 2018;3:366-375.
43. Gussy M, Waters E, Kilpatrick NM. A qualitative study exploring barriers to a model of shared care for pre-school children's oral health. British Dental Journal 2006;201:165-170.
44. Ramazani N. Different Aspects of General Anesthesia in Pediatric Dentistry: A Review. Iranian journal of pediatrics 2016;26:e2613-e2613.

Federal President's Report

Dr Sue Taji

Congratulations are due to all the oral health practitioners who were honoured in the recent Queens Birthday honours list for their selfless dedication to the oral health field. Particular mentions should be made of Professor Richard Welbury CBE who received the award of the Commander of the British Empire as well as Associate/Professor Jamie Lucas AM for receiving the Member of the Order of Australia accolade for their contributions to paediatric dentistry and the oral health of children.

In mid-May, I once again attended the ADA Affiliates meeting in Sydney this year alongside colleagues from other dental fields and specialities and represented our society. A broad spectrum of topics were discussed including various topics of relevance to the field of paediatric dentistry. The continuous communication and collaboration between our society and the Australian Dental Association remains an important link. Such links form essential avenues for our society to have input in establishment of guidelines and policies where required and to do so alongside other groups and affiliates.

At the recent IAPD Council meeting in Cancun, Mexico, Presidents and other formal representatives from the national societies across the globe joined to hear updates provided by the board of the International Association of Paediatric Dentistry (IAPD). The Federal Vice President, Dr Soni Stephen, and I represented the Australia and New Zealand region at the IAPD Council meeting and it is pleasing to see the progress IAPD continues to make in the bettering of the oral health of children, as well as linking the interested societies together to globalise such an initiative. It was encouraging to see that many members from the Australia and New Zealand region attended the IAPD Congress in Cancun, which marked the IAPD's 50th anniversary.

Further to my last report, plans have been set in motion to place a bid for the 2025 IAPD congress, to bring this International congress to our region and to have it held in Melbourne. Dr John Sheahan has been named the chair of the bidding committee. International events of such a calibre bring more great minds within the field of Paediatric Dentistry from far and wide and give colleagues the opportunity to attend a world class congress specific to the field in our region.

Having passed the half way mark in the year, I take this opportunity to remind colleagues of the upcoming ANZSPD Biennial to be held in Hobart in March 2020 and to consider this as you plan the year ahead. Such events continue to provide excellent opportunities to catch up with colleagues from far and wide and enjoy exceptional CPD.

Kind regards,

Dr Sue S. Taji, *ANZSPD President*

IAPD CONGRESS in Cancun, marking IAPD's 50th Anniversary

Brush Happy!

ILLUMINATION PRESENTS DESPICABLE ME MINION MADE™

Colgate®

QUEEN'S BIRTHDAY HONOURS
List Recipients

ANZSPD warmly congratulates everyone honoured in this year's **Queen's Birthday Honours List.** Dr Jamie Lucas was appointed to the Order of Australia in Australia's highest recognition for outstanding achievement and service. Dr Lucas was awarded Member (AM) in the General Division of the Order of Australia for significant service to paediatric dentistry, and to professional organisations.

Congratulations also to Prof. Richard Welbury who was recognised in the 2019 Queen's Birthday Honours and appointed Commander of the British Empire (CBE) for his dedication to safeguarding as well as for his role as both educator and innovator in paediatric dentistry.

ANZSPD congratulates Dr Jillen Patel, for receiving the award of 7News Western Australia Young Achiever of the Year at the 2019 WA Day awards this year.

Dr Patel received the award for his commitment in helping the vulnerable and disadvantaged, supporting families in remote Aboriginal communities, homeles people, refugees in the Perth metropolitan area and children with complex medical needs. He developed the Kimberley Dental Team, providing oral health education and volunteer-based dental services to cohorts across WA.

University of Sydney dental student Kumudika Gunaratne receiving the Louise Brearley Messer Undergraduate Essay prize and certificate from Dr Soni Stephen (Federal Vice President and ANZSPD NSW Councillor) at a function held in Sydney. Congratulations to Mr Gunaratne and the Postgraduate Essay prizewinner, Dr Vanessa Cho from the University of Western Australia.

Nutritive and Non-Nutritive Sucking – a review

Janita Shah, 3nd Year DCD (Paeds)

Introduction

Sucking movements are amongst the earliest coordinated muscle activities to develop during prenatal life. Sucking reflexes develop at approximately 24 weeks in utero, eight to twelve weeks after the emergence of oral and gag reflexes. Feeding on the other hand, which involves a highly complex and energetic suck/swallow/breathe cycle, is not fully coordinated until 32 to 34 weeks[1].

There are two types of sucking - nutritive sucking is related to the process of obtaining nutrition through breast or bottle-feeding, whereas non-nutritive sucking is a habit which involves sucking of digits or objects such as pacifiers/ dummies. These two types vary in duration, rate and strength of sucking. Nutritive sucking occurs at a constant rate of one suck-per-second, whereas non-nutritive sucking occurs at a quicker rate of two sucks-per-second and is thought to satisfy an infant's instinctive sucking urge or serve as a behavioural state modulatory mechanism (comforter)[1].

Nutritive sucking

Nutritive sucking (breast and bottle) may influence craniofacial growth and development, however, the mechanisms of the two forms of feeding differ. Breast-feeding involves squeezing of the mother's nipple by the infant, whereas bottle-feeding involves a piston like action of the tongue when sucking the teat of the bottle2. Additionally, it is believed that breast-feeding and bottle-feeding involve different orofacial muscles and vary in their patterns of muscle activity[3-5]. Therefore, these two forms of feeding are believed to have different effects on craniofacial growth and development.

Breast-feeding has various nutritional, immunological, psychological and developmental benefits[6]. Given these advantages, the World Health Organisation and the NHRMC Infant feeding guidelines have encouraged exclusive feeding for the first six months of life. Prolonged breast-feeding has a protective effect on the development of malocclusion, resulting in lower rates of anterior open-bites and posterior cross-bites[4,5,7-10]. Meanwhile other studies did not find an association between breast-feeding and development of malocclusions[11,12].

This protective influence of breast-feeding on the development of malocclusion can be explained by the following: the absence of a continuous flow of milk during breast-feeding places greater demands on the infant's orofacial muscles, subsequently encouraging growth and development of the mandible and surrounding musculature[13]. There is sufficient evidence to state that longer duration of breast-feeding can reduce the risk of developing non-nutritive sucking habits such as pacifier sucking[14-16]. It is believed that breast-feeding provides a greater sense of fulfilment and security which satisfies the instinctive sucking needs of infants, such that they are less likely to engage in non-sucking behaviours. On the other hand, the large bottle teat and resultant increased milk flow may lead to a preference for the pacifier as the infant grows[13,16-19].

A systematic review of cohort studies could not confirm the types of malocclusion associated with bottle-feeding, or the optimal duration of exclusive breast-feeding to protect against malocclusion. The authors hypothesised that the presence of confounding variables such as non-nutritive sucking habits may have distorted the results and that more research is required to further explore these associations20. Until then, exclusive breast-feeding for at least six months, as per the WHO21 and NHRMC infant feeding guidelines22, is still the best recommendation to benefit children in terms of their systemic health.

Non-nutritive sucking

Nearly all infants engage in some type of habitual non-nutritive sucking. In most cases this refers to pacifier or digit sucking. However, other objects such as toys and blankets may also be involved23, 24. Foetuses have been reported to suck their thumbs in utero, and infants have been reported do so from six months to two years, or later. Pacifier use has greatly increased in recent times with a corresponding decrease in digit sucking25-27, and it is rare for an infant to engage in both habits28. To some extent, the practice of non-nutritive sucking is culturally determined since children who are allowed ready access to the mother's breast for an extended period, rarely suck any other object 24, 28.

Non-nutritive sucking presents many benefits to healthy and preterm infants, namely, helping calm infants, helping them to sleep and stop crying, helping reduce pain during painful minor procedures in the emergency department (e.g. venepuncture)29, 30 as well as being associated with a lower risk of sudden infant death syndrome (SIDS)31, 32.33-35. Sudden infant death syndrome is defined as the sudden death of an infant that was unexpected by history and unexplained by a post-mortem examination that includes a case investigation, complete autopsy, and examination of the death scene34. Three mechanisms have been proposed as to why pacifier use may be protective against SIDS. Pacifiers are thought to maintain airway patency during sleep by preventing the backward positioning of the tongue, they may reduce gastric reflux, and may stimulate respiration thereby reducing apnoeic episodes36. Therefore, pacifiers have been recommended in infants up to 1 year-of-age once breast-feeding has been well established, which includes the peak ages for sudden infant death syndrome risk and the period in which the infant's need for sucking is highest34.

There are some clinical benefits of non-nutritive sucking in preterm infants whose ability to feed is under-developed and therefore require feeding tubes initially. Pacifier use promotes the development of sucking in preterm infants, allowing a more rapid transition from tube to

bottle feeds and better bottle-feeding performance. Additionally, pacifier use may also help digestion by stimulating vagal nerve innervation in the oral mucosa which subsequently increases enzyme production such as lipase, insulin and motilin. These clinical benefits reduce the duration of hospital stay in preterm infants[37].

Non-nutritive sucking decreases in prevalence as the child grows older[28]. Pacifier sucking decreases rapidly from infancy and usually stops by four years once interaction with other children increases[23, 24, 28]. Thumb-sucking also decreases until about four years of age after which the prevalence plateaus until seven years before once again declining28. However, a small proportion of children may continue thumb-sucking after the age of eight. Pacifier sucking is considered to be an easier habit to stop due to the ability to remove a pacifier from the child[24, 38].

Although sucking has proven to be beneficial, especially in the first two years of life, prolonged non-nutritive sucking if extended beyond the age of three or four years may be associated with a range of adverse effects on oro-facial development. Pacifier use has also been associated with increased episodes of otitis media, cough, wheezing, diarrhoea/gastroenteritis, latex allergy and increased oral colonisation with Candida in infants. Similarly, digit sucking may result in intestinal parasitic infections, digital deformity and paronychia[31, 36].

Effects of non-nutritive sucking habits on the developing dentition.

Non-nutritive sucking is common, however, malocclusions are only noted in a small proportion of children who engage in such habits. The effects on the primary and permanent dentition depends on the duration, frequency, magnitude and intensity of the habit. Dental manifestations may include anterior open-bite, posterior cross-bite, increased overjet, and a higher tendency towards developing a Class II dental relationship[14].

Physiological or orthodontic pacifiers have been available since the 1990s and are designed to conform better to the child's intraoral structures and to produce a more physiologic sucking pattern thus limiting adverse effects on orofacial development[23, 39, 40]. However, the limited number of studies assessing the impact of these specially designed pacifiers on the developing occlusion have found little benefit when compared with conventional pacifiers or digit sucking[41].

Pacifier and digit sucking have been reported to have different effects on the dental development. Prolonged pacifier use has been linked to anterior open-bite, Class II molar relationships, and posterior cross-bite, whereas digit sucking has been associated with anterior open-bite and increased overjet[11, 24]. Pacifier-associated anterior open-bite is usually symmetrical in nature with the maxillary incisors in close proximity to each other, due to the pacifier's shape and form which limits its positioning in the mouth[24]. On the other hand, digit sucking results in the development of an asymmetric open-bite as dictated by the position of the digit in the mouth, with the maxillary incisors proclined and spaced. Severe cases of digit sucking can also cause retrusion of the mandibular incisors further worsening the overjet[23].

Pacifier use has been associated with a narrowing of maxillary inter-canine and inter-molar widths resulting in posterior crossbite[23, 28]. During pacifier sucking, the tongue's lower positioning can lead to dento-alveolar expansion of the mandibular arch. The increased contraction of the buccinator muscles during pacifier sucking can exert narrowing forces against the maxillary arch form resulting in a constricted or narrow maxilla[23, 24]. A posterior cross-bite can develop as early as 18 months. If a functional unilateral cross-bite develops, the deviation of the mandible upon closure may affect mandibular development[24].

Pacifier use has a more consistent impact on both the anterior and posterior occlusions; although, due to the comparatively shorter duration of pacifier use, its effect on the mixed and permanent dentition may be less when compared to prolonged digit sucking[24, 42].

Duration of the habit can mean either duration (years) during which the habit actively occurs or overall time (hours per day) that the child engages in the habit. Few studies have investigated the impact of the former, however, habits of less than six hours duration per day are unlikely to have an impact on oro-facial development because orthodontic forces of shorter duration are usually insignificant23. Pacifier sucking for greater than two years and digit sucking for greater than three years is associated with significantly higher rates of posterior cross-bite and anterior open-bite in the primary dentition by the age of five years[11]. Of greater concern, however, is the impact of non-nutritive sucking on the mixed, and especially permanent dentitions. Engaging in non-nutritive sucking habits for more than three years was reported to significantly increase the risk of malocclusion in the mixed dentition[43]. Those who used pacifiers beyond the age of four had a higher risk of developing an anterior open-bite during the mixed dentition stage and Class II molar relationships. Children with digit sucking habits persisting for longer than six years had a higher chance of developing anterior open-bites[43].

Generally, dental malocclusion due to non-nutritive sucking tends to improve once the habit is stopped. However, this can take two to five years to completely resolve and it is dependent on several factors such as growth pattern, overall duration of the habit, and presence of other coexisting habits such as tongue thrusting[44, 45]. The type of occlusion is also improtant, with posterior cross-bites more resistant to self-resolution when compared to anterior open-bites[24, 45]. As long as the habit stops by the age of six years, most effects on the dentition are transient and likely to resolve spontaneously by the age of eight to 12 years[46]. Therefore, although the American Academy of Paediatric Dentistry's policy on oral habits recommend that habit cessation should be encouraged by three years[47], and others recommend decreasing pacifier use by age two and discontinuation by age four[48], the critical time appears to be six years, after which the spontaneous correction of an associated malocclusion is unlikely.

Management of non-nutritive sucking habits

The literature is inconclusive on the ideal age to start habit cessation treatment, with some authors suggesting no intervention is necessary as most children tend to discontinue the habits before the permanent teeth begin to erupt and any effects on the dental development at this stage is transient and usually resolves upon cessation of the habit49. Others recommend some form of intervention if the child is still engaging in the habit beyond the age of three years[43]. A recent Cochrane review concluded that psychological intervention and appliance therapy are more effective at ceasing a non-nutritive sucking habit when compared to no intervention[50]. However, no conclusions could be made regarding whether psychological intervention

on its own was more effective than appliance therapy on its own or whether the two together provided better clinical outcomes[50]. Generally non-invasive methods such as positive reinforcement and rewards should be attempted first, and more invasive options such as appliance therapy are only indicated if all other avenues have been exhausted and the child is continuing the habit into the mixed dentition stage[23].

There is no standard intervention for cessation of non-nutritive sucking habits. Various approaches and interventions have been described and attempted with varying degrees of success ranging from behaviour modification, to application of adverse tasting medicaments to the digit, to fitting an appliance that prevents the comfortable seating of the digit or object in the mouth[23]. Treatment is directed towards decreasing or eliminating the habit and minimizing potential deleterious effects on the dentofacial complex. Early dental visits have been recommended to allow assessment of infants engaging in such behaviours and to provide anticipatory guidance to the parents[47].

Positive reinforcement and rewards

The simplest approach to habit therapy is a straightforward explanatory discussion between the child and the dentist that expresses concern regarding the habit. This "adult approach" is often enough to stop the habit in more mature children23. At home, positive encouragement and consistent reminders for the child to stop the habit. Parents should adopt a positive approach when encouraging habit cessation as negative reinforcement and punishment may result in worsening of the habit[51]. The use of a reward system can be implemented that provides a small tangible reward for every day that the child does not engage in the habit. In some cases, a larger reward can be negotiated for complete cessation of the habit. A calendar system can be useful to keep track of the child's progress towards habit cessation[23]. Usually, if the child can abstain from the habit for at least three months, it is likely to indicate cessation[23, 51].

Response Prevention therapy

Response prevention therapy is aimed to discourage a child from engaging in non-nutritive sucking habits by either physically preventing them from placing digits or other objects in their mouth or by placing an unpleasant taste on the offending digit or object. This acts as a reminder to a child who is motivated and willing to try and stop the habit and can be recommended as an alternative or adjunct to positive reinforcement and rewards. Products such as thumb guards, bandages, gloves, mittens and spicy or bitter-tasting medicaments have been found to be effective in stopping both day time and night time sucking habits[23, 52, 53]. These products are easily available, easy to use and inexpensive, however, they are also easy to remove and ignore by children with a more ingrained habit, who are reluctant to stop[54]. In this small proportion of children who continue engaging in non-nutritive habit into their mixed dentition stage, appliance therapy is often indicated[23, 51].

Appliance therapy

When all other attempts to stop non-nutritive sucking habits have failed, appliance therapy is often indicated. It is important that the child understands that the appliance is not punishment for continuing the habit, but rather a reminder for the child who is keen to stop. Lack of compliance can result in failure, acquisition of new compensatory habits (e.g. nail-biting, scratching of the body or knuckle cracking), deformation or early removal of the appliance[23, 51, 55]. Children with a high caries risk, poor oral hygiene and intellectual disability are unsuitable for appliance therapy[54]. Appliances may be either removable or fixed. Removable appliances, such as a Hawley retainer with a series of loops palatal to the incisors, however, the child is able to remove the appliance whenever they want[51, 56].

Palatal Crib

Palatal cribs prevent the comfortable positioning of digits or objects against the palate and the child no longer finds digit or pacifier sucking pleasurable. They also limit any associated tongue thrust habit that has developed and allow the natural force of the lips to correct the anterior open-bite23, 51, 55, 57. The palatal crib can be cemented to either the first permanent molars or the second primary molars. The appliance consists of a major connector wire (0.04" stainless steel wire) which extends anteriorly along the palate. Adjacent to the maxillary canines, the wire is bent to form a "fence" or a "crib" which extends vertically downwards, behind the incisor teeth, to the level of the incisal edges of the mandibular incisors. The appliance should not interfere with occlusion and there should be sufficient clearance to allow for retroclination of the proclined upper incisors [23, 51]. Rakes and spurs can also be added, however, these may be unnecessarily punitive[55, 58].

High success rates of palatal cribs have been reported with four out of five children stopping thumb sucking within one week of placement and little relapse at three-year follow-up[55, 57, 59]. Lower chances of relapse were noted if palatal cribs were retained in place for at least six months and higher relapse rates noted if the appliance was removed within three months of placement[57]. Along with cessation of the habit, improvement of teeth alignment has been observed after three months of insertion of the appliance, and correction of the anterior open-bite within six months[51]. However, correction of the malocclusion is also dependent on other factors such as presence of other coexisting habits and the dental maturity of the child[51].

A quad-helix with a crib attachment may be indicated in children who have developed a dentoalveolar anterior open bite and posterior crossbite in the mixed dentition due to continued digit sucking. This modification to the palatal crib not only stops the habit but also corrects the malocclusion[59-61]. It further improves skeletal relationships by significantly increasing the overbite as the permanent incisors are extruded and the palatal plane is rotated downwards[59].

There are disadvantages associated with the use of palatal cribs. Initially, children often find it difficult to masticate and swallow with the appliance in situ. However, this is usually a transient issue and they eventually adapt to the presence of the appliance. Also, after placement, speech problems such as slurred or lisped speech may develop, but this usually resolves after the appliance is removed and sometimes even during active treatment stage[55]. There have been reports of mechanical irritation to the palatal mucosa caused by poorly designed or fabricated appliances [51, 55]. Loosening or loss of the appliance also occurs occasionally [54].

Bluegrass appliance

Introduced in 1991, this appliance is considered a non-punitive alternative for managing chronic digit sucking[58]. It is thought to provide the child with a "toy" that they can roll with their tongue so that they are distracted from digit sucking, thereby acting as a counter

conditioning response to the original conditioned stimulus for digit sucking[58, 62]. There are several designs of this appliance although the basic design is a small barrel-shaped six-sided Teflon-coated roller or multiple colourful beads threaded over a 0.045" stainless steel wire which is soldered to molar bands which are cemented either to primary second molars or permanent first molars. The roller or beads are placed at the highest point of the palate and must be free to be rolled when moved by the tongue[58]. The Bluegrass appliance has reported to stop a digit sucking habit within three months[62]. Advantages of this appliance include better acceptance by children and their parents and fewer effects on speech and mastication[58]. The Bluegrass appliance has also been incorporated into a quad-helix, with an additional crib attachment in a fixed-removable design that allows easy activation of the quad-helix after insertion[63].

Psychological intervention

If the habit persists despite attempting all the above-mentioned management options, consideration should be given to the psychopathological cause of the habit, and the child or adolescent should be referred to an appropriate psychologist or medical practitioner for assessment[23, 51].

Conclusion

Nutritive and non-nutritive sucking habits are among the most commonly reported oral habits in children. The role of nutritive sucking on craniofacial development is not yet clearly proven. However, prolonged non-nutritive sucking habits have been shown to result in malocclusion in the primary and permanent dentition. Cessation of non-nutritive sucking habits prior to the eruption of the permanent dentition is essential for achieving spontaneous resolution of malocclusion. Dental professionals who care for children should be able to provide anticipatory guidance to families, ensure timely detection of sucking habits, and be aware of the tools available to encourage habit cessation.

References

1. Harding C. The use of nonnutritive sucking to facilitate oral feeding in a term infant: a single case study. Journal of pediatric nursing 2012;27(6):700.
2. Viggiano D, Fasano D, Monaco G, Strohmenger L, Breast feeding, bottle feeding, and non-nutritive sucking; effects on occlusion in deciduous dentition. 2005.
3. Wang XT, Ge LH. [Influence of feeding patterns on the development of teeth, dentition and jaw in children]. Beijing da xue xue bao Yi xue ban = Journal of Peking University Health sciences 2015;47(1):191-5.
4. Romero CC, Scavone-Junior H, Garib DG, Cotrim-Ferreira FA, Ferreira RI. Breastfeeding and non-nutritive sucking patterns related to the prevalence of anterior open bite in primary dentition. Journal Of Applied Oral Science: Revista FOB 2011;19(2):161-8.
5. Sánchez-Molins M, Grau Carbó J, Lischeid Gaig C, Ustrell Torrent JM. Comparative study of the craniofacial growth depending on the type of lactation received. European Journal Of Paediatric Dentistry: Official Journal Of European Academy Of Paediatric Dentistry 2010;11(2):87-92.
6. Kramer MS, Kakuma R. Optimal duration of exclusive breastfeeding. Cochrane Database of Systematic Reviews 2012;(8).
7. Kobayashi HM, Scavone Jr H, Ferreira RI, Garib DG. Relationship between breastfeeding duration and prevalence of posterior crossbite in the deciduous dentition. 2010.
8. Peres KG, Barros AJD, Peres MA, Victora CG. Effects of breastfeeding and sucking habits on malocclusion in a birth cohort study. Revista De Saude Publica 2007;41(3):343-50.
9. Moimaz Suzely A, Zina L, Saliba N, Saliba O. Association between breast-feeding practices and sucking habits: A cross-sectional study of children in their first year of life. Journal of Indian Society of Pedodontics and Preventive Dentistry, Vol 26, Iss 3, Pp 102-106 (2008) 2008;(3):102.
10. Caramez da Silva F, Justo Giugliani ER, Capsi Pires S. Duration of breastfeeding and distoclusion in the deciduous dentition. Breastfeeding Medicine: The Official Journal Of The Academy Of Breastfeeding Medicine 2012;7(6):464-8.
11. Warren JJ, Bishara SE. Original Article: Duration of nutritive and nonnutritive sucking behaviors and their effects on the dental arches in the primary dentition. American Journal of Orthodontics & Dentofacial Orthopedics 2002;121:347-56.
12. Luz CLF GD, Arouca R. Association between breast-feeding duration and mandibular retrusion: a cross sectional study of children in the mixed dentition. American Journal of Orthodontics & Dentofacial Orthopedics 2006;130:531-4.
13. Charchut SW, Allred EN, Needleman HL. The effects of infant feeding patterns on the occlusion of the primary dentition. Journal of dentistry for children (Chicago, Ill) 2003;70(3):197-203.
14. Silva M, Manton D. Oral Habits–Part 1: The Dental Effects and Management of Nutritive and Non-nutritive Sucking. Journal of Dentistry for Children 2014;81(3):133-9.
15. Lopes-Freire GM, Cardenas AB, Suarez de Deza JE, Ustrell-Torrent JM, Oliveira LB, Boj Quesada JR, Jr. Exploring the association between feeding habits, non-nutritive sucking habits, and malocclusions in the deciduous dentition. Progress in orthodontics 2015;16:43.
16. Narbutytė I, Narbutytė A, Linkevičienė L. Relationship between breastfeeding, bottle-feeding and development of malocclusion. Stomatologija 2013;15(3):67-72.
17. Montaldo L, Montaldo P, Cuccaro P, Caramico N, Minervini G. Effects of feeding on non-nutritive sucking habits and implications on occlusion in mixed dentition. Int J Paediatr Dent 2011;21(1):68-73.
18. Scavone-Jr H, H Guimarães-Jr C, Ferreira R, Nahás-Scocate A, Vellini-Ferreira F, Association between breastfeeding duration and non- nutritive sucking habits. 2008.
19. Lopes Mesquita Freire G, Carolina Linhares Ferrari J, Percinoto C, Association between maternal breastfeeding and the development of non-nutritive sucking habits. 2015.
20. Hermont AP, Martins CC, Zina LG, Auad SM, Paiva SM, Pordeus IA. Breastfeeding, bottle feeding practices and malocclusion in the primary dentition: a systematic review of cohort studies. International journal of environmental research and public health 2015;12(3):3133-51.
21. Organization WH, Infant and young child nutrition. Global strategy on infant and young child feeding., 2002.
22. Council) NNHaMR, Infant Feeding Guidelines: Summary. in Council NHaMR, ed. Canberra, Australia, 2013.
23. Proffit WR, Fields HW, Contemporary Orthodontics. Mosby-Year Book, 1993.
24. KAREN D, CLARE M, J. IA, R. SJ. Sucking habits in childhood and the effects on the primary dentition: findings of the Avon Longitudinal Study of Pregnancy and Childhood. International Journal of Paediatric Dentistry 2008;18(3):178-88.
25. D. BB. A longitudinal study of digital and dummy sucking*. Australian dental journal 1966;11(3):184-90.
26. Backlund E. Facial growth, and the significance of oral habits, mouthbreathing and soft tissues for malocclusion. A study on children around the age of 10. Acta odontologica Scandinavica 1963;21:Suppl 36:9139.
27. Larsson EF, Dahlin KG. The prevalence and the etiology of the initial dummy- and finger-sucking habit. Am J Orthod 1985;87(5):432-5.
28. Bishara SE, Warren JJ, Broffitt B, Levy SM. Changes in the prevalence of nonnutritive sucking patterns in the first 8 years of life. American Journal of Orthodontics and Dentofacial Orthopedics 2006;130(1):31-6.
29. Zempsky WT, Cravero JP. Relief of pain and anxiety in pediatric patients in emergency medical systems. Pediatrics 2004;114(5):1348-56.
30. Curtis SJ, Jou H, Ali S, Vandermeer B, Klassen T. A randomized controlled trial of sucrose and/or pacifier as analgesia for infants receiving venipuncture in a pediatric emergency department. BMC pediatrics 2007;7:27.
31. SC HR, R HF, Y MR, P LHM, S BP. Dummy (pacifier) use and sudden infant death syndrome: Potential advantages and disadvantages. Journal of Paediatrics and Child Health 2014;50(3):170-4.
32. Pollard K, Fleming P, Young J, Sawczenko A, Blair P. Night-time non-nutritive sucking in infants aged 1 to 5 months: relationship with infant state, breastfeeding, and bed-sharing versus room-sharing. Early Human Development 1999;56(2):185-204.
33. Woodson R, Drinkwin J, Hamilton C. Effects of nonnutritive sucking on state and activity: Term-preterm comparisons. Infant Behavior and Development 1985;8(4):435-41.
34. Hauck FR, Omojokun OO, Siadaty MS. Do Pacifiers Reduce the Risk of Sudden Infant Death Syndrome? A Meta-analysis. Pediatrics 2005;116(5):e716.
35. Fleming PJ, Blair PS, Bacon C, Bensley D, Smith I, Taylor E, et al. Environment of infants during sleep and risk of the sudden infant death syndrome: results of 1993-5 case-control study for confidential inquiry into stillbirths and deaths in infancy. BMJ 1996;313(7051):191-5.
36. Cinar DN. The advantages and disadvantages of pacifier use. Contemporary nurse 2004;17(1-2):109-12.
37. Pinelli J, Symington A. Non-nutritive sucking for promoting physiologic stability and nutrition in preterm infants. The Cochrane database of systematic reviews 2005;(4):CD001071.
38. Cerny R. Thumb and finger sucking. Australian

References continued on page 16.

Premature eruption and severe enamel hypoplasia in successor premolars subsequent to caries and peri-radicular infection in primary molars: a review and report of two cases

Dr Amanda Leske
Doctor of Clinical Dentistry (Paediatric Dentistry) Candidate, The University of Melbourne

Abstract

Severe caries leading to peri-radicular infection and inflammation in primary teeth is known to cause developmental defects in their permanent successors. However, there is very little evidence to suggest which teeth might be most susceptible to such complications. This case report describes two paediatric patients who experienced significant developmental defects in their permanent teeth subsequent to caries-related infection in the primary predecessors. This report highlights factors that might indicate children are at risk of such events, in order to better guide treatment planning for patients with early childhood caries.

Introduction

It is well known that pulpal infection of primary teeth and subsequent peri-apical inflammation and infection may lead to developmental defects in the successor permanent teeth. This has been demonstrated clinically, radiographically, experimentally in animals and histologically from human autopsy material over several investigations[1-9]. Reported deleterious effects of primary tooth infection on its permanent successor include enamel hypoplasia, enamel hypomineralisation, tooth discolouration, cyst formation, delayed or premature eruption, ectopic eruption, arrest of root development, dilaceration of crown/root, bone loss and sequestration of the tooth germ [3, 4, 9-14].

Turner was one of the first to describe a spectrum of enamel hypoplasia ranging from mild discolouration to severe crown dysmorphia that was isolated to a single permanent tooth associated with pathology in its primary predecessor; subsequently, such developmental defects came to be known as Turner's hypoplasia, or Turner's teeth[2.] When found on an anterior permanent tooth, enamel hypoplasia is most likely due to a previous traumatic incident involving the overlying primary tooth[15.] Conversely, premolar teeth are most commonly affected by enamel hypoplasia secondary to infection in the overlying primary tooth, due to the relatively higher caries prevalence of primary molars[5]. Four main categories of Turner's hypoplasia were identified by Silberman et al.[16] (Table 1). Permanent teeth affected by enamel hypoplasia can present significant clinical challenges in terms of preventing plaque retention and dental caries, restoring aesthetics, function, occlusion and preserving pulpal health[5].

It is clear that not every infected primary tooth will result in altered development of the permanent successor. The chances of an infection in a primary tooth leading to developmental defects in the underlying permanent tooth is likely a culmination of multiple factors and is estimated to occur in only one quarter to one fifth of cases 1, 3. There is currently little evidence to help determine the level of risk to the permanent successor from an infected primary tooth. Therefore, it is important to report cases where significant anomalies of permanent teeth have resulted from pathology in primary teeth, to better inform parents, clinicians and organisations when making decisions regarding treatment options and timing of treatment of infected primary teeth. This report describes two cases of significant developmental disturbances in permanent teeth as a consequence of dental caries and pulpal infection in the overlying primary teeth.

Case One

A six-year-old girl with Latino heritage and no relevant medical history presented to the Paediatric Dentistry Department at The Royal Dental Hospital Melbourne with pain on eating from the lower left quadrant. The patient had a history of attendance at the hospital's emergency dental department due to pain from tooth 75 three years prior. Subsequently, when the patient was three years and ten months of age, dental treatment was provided under general anaesthesia (GA) including uncomplicated extraction of severely carious teeth 75 and 85, restoration of tooth 65 and fissure sealant placement on teeth 55, 54, 64, 74 and 84. An orthopantomogram (OPG) from the initial consultation and bitewing radiographs taken at the time of GA are shown in Figures 1 and 2. Of note is the bony rarefaction evident in the bifurcation

Table 1. *Types of enamel hypoplasia. Adapted from Silberman et al.* [16]

Type I hypoplasia	Enamel discoloration due to hypoplasia
Type II hypoplasia	Abnormal coalescence due to hypoplasia
Type III hypoplasia	Some parts of enamel missing due to hypoplasia
Type IV hypoplasia	A combination of types I, II and III

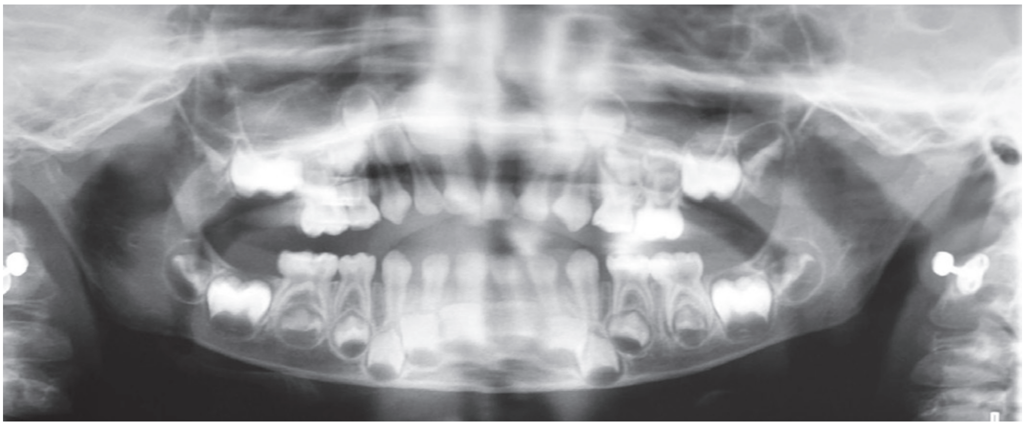

Figure 1. *Orthopantomogram taken at initial consultation at three years and eight months-of-age demonstrating caries in teeth 85 and 75 and bifurcation radiolucency associated with tooth 75*

region of tooth 75 and the abnormal follicular outline of developing tooth 35. Following dental treatment under GA, the patient was referred for follow-up reviews in the Paediatric Dentistry Department, however, failed to attend review appointments until the current presentation.

On examination, the patient was found to be in an age-appropriate early mixed dentition apart from the presence of a prematurely erupted tooth 35, which demonstrated severe enamel hypoplasia with brown discolouration that had exposed a large amount of dentine which had become carious on multiple surfaces. The tooth was tender on percussion with grade one mobility. Cavitated carious lesions extending into dentine were also diagnosed in teeth 55, 54, 64, 65, 26, 36 and 46, which were confirmed on bitewing radiographs (see Figure 3), with enamel caries noted on tooth 74 distal surface. An OPG (see Figure 4) confirmed the severe enamel hypoplasia of tooth 35, severely dysmorphic crown and root structure and a significant peri-radicular radiolucency. Tooth 35 was designated a poor prognosis and planned for extraction, along with restoration of carious teeth as appropriate and remineralisation of tooth 74. The patient was not able to tolerate dental treatment in the dental chair and a second GA was deemed necessary.

Under GA two months later, when the patient was six years and eight months-of-age, a full examination, dental prophylaxis and fluoride application (Duraphat®, Colgate Oral Care, Sydney, NSW, Australia) was performed, a fissure sealant (ClinPro™, 3M™ESPE™, NSW, Australia) was placed on tooth 16, preformed metal crowns (3M™ESPE™, NSW, Australia) were fitted on teeth 55, 54, 64 and 65 after carious tissue removal and conventional crown preparation and resin-based composite restorations (Tetric N-Ceram, Ivoclar Vivadent, Schaan, Liechtenstein) were placed in teeth 26, 36 and 46 following carious tissue removal. Local analgesia (2 mL lignocaine 2% 1:80 000 adrenaline) was administered via buccal submucosal infiltration followed by simple elevation of tooth 35 which was subsequently preserved in 10% buffered formalin and sent for histopathological examination. After decalcification and staining in haematoxylin and eosin (H&E), microscopy revealed a non-vital, carious, dysmorphic and under-developed premolar tooth with thin enamel matrix space suggestive of hypoplastic enamel (see Figure 5). Large

References continued from page 14.

dental journal 1981;26(3):167-71.
39. Zardetto C, Regina Martins Delgado Rodrigues C, Stefani F, Effects of different pacifiers on the primary dentition and oral myofunctional strutures of preschool children. 2002.
40. Zimmer S, Barthel CR, Ljubicic R, Bizhang M, Raab WH. Efficacy of a novel pacifier in the prevention of anterior open bite. Pediatr Dent 2011;33(1):52-5.
41. Nelson AM. A Comprehensive Review of Evidence and Current Recommendations Related to Pacifier Usage. Journal of Pediatric Nursing 2012;27(6):690-9.
42. Dimberg L, Lennartsson B, Söderfeldt B, Bondemark L. Malocclusions in children at 3 and 7 years of age: a longitudinal study. European Journal of Orthodontics 2013;35(1):131-7.
43. Warren J, Slayton R, E Bishara S, M Levy S, Yonezu T, Kanellis M, Effects of non-nutritive sucking habits on occlusal characteristics in the mixed detition. 2005.
44. D. BB. The effects of digital and dummy sucking on arch widths, overbite, and overjet: A longitudinal study*. Australian dental journal 1966;11(6):396-404.
45. Heimer MV, Tornisiello Katz CR, Rosenblatt A. Non-nutritive sucking habits, dental malocclusions, and facial morphology in Brazilian children: a longitudinal study. European Journal of Orthodontics 2008;30(6):580-5.
46. Popovich F, Thompson GW. Thumb- and finger-sucking: Its relation to malocclusion. American Journal of Orthodontics 1973;63(2):148-55.
47. Guideline on Management of the Developing Dentition and Occlusion in Pediatric Dentistry. Pediatric Dentistry 2015;37(6):253-65.
48. Adair SM. Pacifier use in children: a review of recent literature. Pediatr Dent 2003;25(5):449-58.
49. Burford D, Noar JH. The Causes, Diagnosis and Treatment of Anterior Open Bite. Dental Update 2003;30(5):235-41.
50. Borrie FRP, Bearn DR, Innes NPT, Iheozor-Ejiofor Z. Interventions for the cessation of non-nutritive sucking habits in children. Cochrane Database of Systematic Reviews 2015;(3).
51. Dean JA, Jones JE, Vinson LAW, McDonald RE, McDonald and Avery's dentistry for the child and adolescent. Elsevier ClinicalKey Australia. St. Louis, Missouri : Elsevier, [2016]
Tenth edition., 2016.
52. Lassen MK, Fluet NR. Elimination of nocturnal thumbsucking by glove wearing. Journal of Behavior Therapy and Experimental Psychiatry 1978;9(1):85.
53. Ross JA. Parents modify thumbsucking: A case study. Journal of Behavior Therapy and Experimental Psychiatry 1975;6(3):248-9.
54. AlEmran S. A new method in reminder therapy technique for ceasing digit sucking habit in children. Journal of Clinical Pediatric Dentistry 2000;24(4):261-3.
55. Haryett RD, Hansen FC, Davidson PO. Chronic thumb-sucking: A second report on treatment and its psychological effects. American Journal of Orthodontics 1970;57(2):164-78.
56. Giuntini V, Franchi L, Baccetti T, Mucedero M, Cozza P, Dentoskeletal changes associated with fixed and removable appliances with a crib in open-bite patients in the mixed dentition. 2008.
57. Haryett RD, Hansen FC, Davidson PO, Sandilands ML. Chronic thumb-sucking: The psychologic effects and the relative effectiveness of various methods of treatment. American Journal of Orthodontics 1967;53(8):569-85.
58. Haskell B, R Mink J, An aid to stop thumb sucking: the "Bluegrass" appliance. 1991.
59. Cozza P, Baccetti T, Franchi L, A McNamara J, Treatment effects of a modified quad-helix in patients with dentoskeletal open bites. 2006.
60. Cozza P, Giancotti A, Rosignoli L, Use of a modified Quad Helix in early interceptive treatment. 2000.
61. Cozza P, Mucedero M, Baccetti T, Franchi L. Treatment and Posttreatment Effects of Quad-Helix/Crib Therapy of Dentoskeletal Open Bite. The Angle Orthodontist 2007;77(4):640-5.
62. Greenleaf S, Mink J. A retrospective study of the use of the Bluegrass appliance in the cessation of thumb habits. Pediatr Dent 2003;25(6):587-90.
63. Kulkarni G, Lau D, A Single Appliance for the Correction of Digit-sucking, Tongue-thrust, and Posterior Crossbite. 2009.

amounts of abnormal, mostly atubular secondary dentine was noted, with pulp stones included in the pulp space. The pulp was noted to be necrotic with an associated peri-radicular granuloma.

The four-week post-operative review demonstrated good oral hygiene, sound restorations and good healing of tooth 35 extraction socket (see Figure 5). Ongoing reviews and fluoride varnish applications are planned on a three-monthly basis, with monitoring of growth and development until the patient is at an appropriate age and level of compliance to treatment plan for the missing tooth 35.

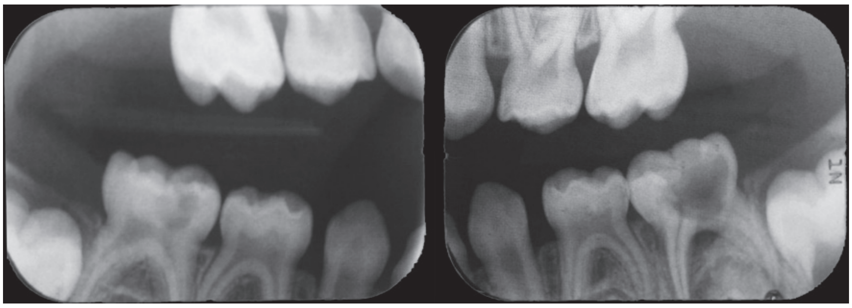

Figure 2. *Bitewing radiographs taken at the time of general anaesthesia at three years and ten months-of-age demonstrating severe caries of teeth 85 and 75, radiolucency at the bifurcation of tooth 75 and abnormal appearance of the follicle surrounding the succedaneous tooth 35*

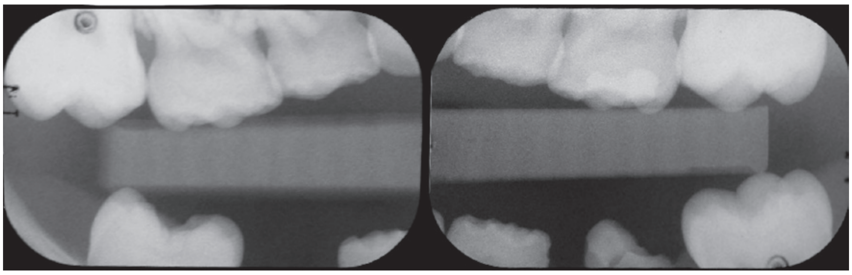

Figure 2. *Bitewing radiographs taken at consultation at six years and five months-of-age demonstrating the prematurely erupted and dysmorphic tooth 35 along with multiple carious lesions in other primary and permanent teeth*

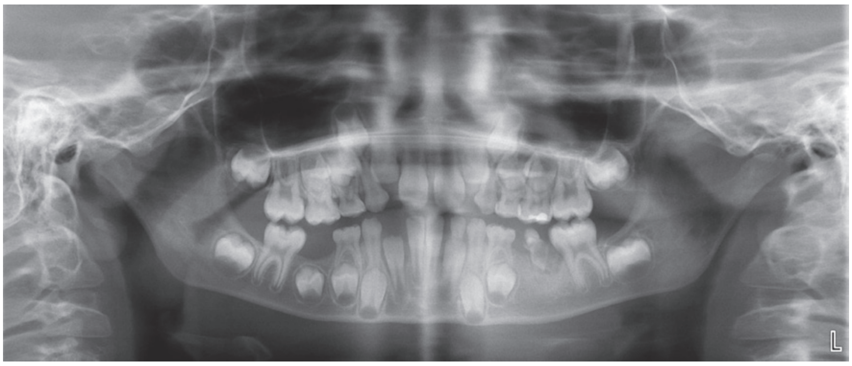

Figure 4. *Orthopantomogram taken at consultation prior to the second general anaesthesia for dental treatment at six years and five months-of-age demonstrating premature eruption of tooth 35 with severe enamel hypoplasia and dysmorphic tooth structure, arrested root development and peri-radicular radiolucency*

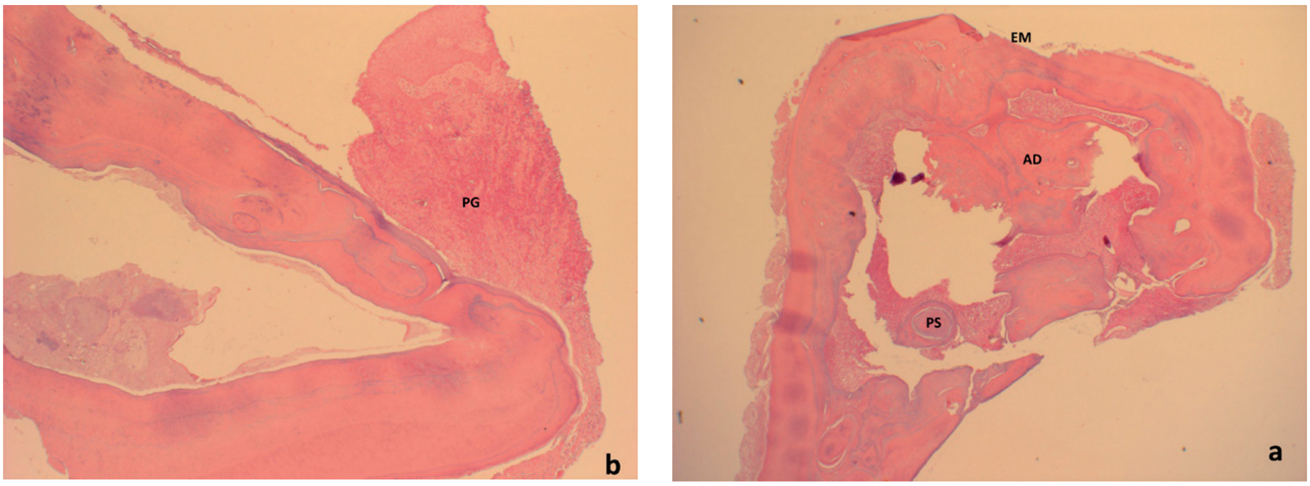

Figure 5. *Photomicrographs of the extracted tooth 35, H & E staining. (a) Section of crown showing haphazard atubular dentine (AD), thin enamel matrix (EM) suggestive of enamel hypoplasia and necrotic pulp space containing bacteria, inflammatory cells and pulp stones (PS) (b) peri-apex area demonstrating necrotic pulp space containing bacteria and inflammatory cells with associated peri-radicular granuloma (PG)*

Figure 6. *Intra-oral photographs taken at the post-operative review four weeks following dental treatment under general anaesthesia demonstrating satisfactory soft tissue healing in the 35 extraction site, good oral hygiene and sound restorations*

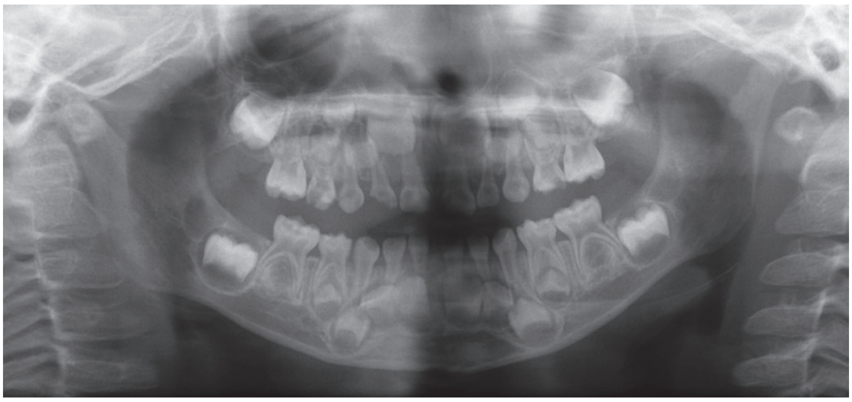

Figure 7. *OPG taken at initial consultation when the patient was three years and four months-of-age demonstrating severe caries in multiple primary teeth including teeth 54 and 64 with the follicles of successor teeth 14 and 24 intimately associated with the furcation area*

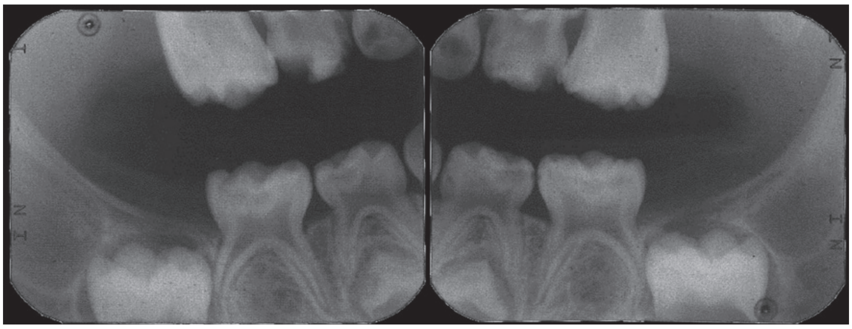

Figure 8. *Bitewing radiographs taken at the time of general anaesthesia at three years and ten months-of-age demonstrating severe caries of teeth 85 and 75, radiolucency at the bifurcation of tooth 75 and abnormal appearance of the follicle surrounding the succedaneous tooth 35*

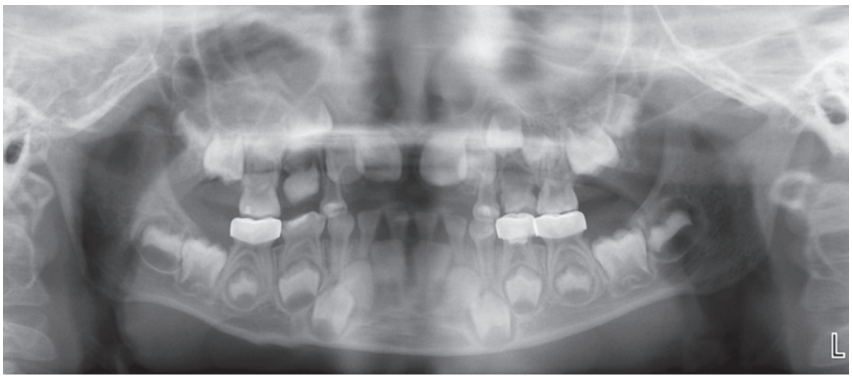

Figure 9. *Bitewing radiographs taken at consultation at six years and five months-of-age demonstrating the prematurely erupted and dysmorphic tooth 35 along with multiple carious lesions in other primary and permanent teeth*

Case Two

A six-year-old boy of Chinese heritage with latex intolerance but otherwise no relevant medical history, attended the Paediatric Dentistry Department, with pain on eating from teeth 14 and 24. The patient had a history of attendance at the hospital's emergency dental department due to pain from upper primary molars three years prior. Subsequently, when the patient was three years and six months-of-age, dental treatment was provided under GA including an MTA pulpotomy on tooth 74, restoration of teeth 55, 53, 63, 65, 74, 75, 83 and 85 and uncomplicated extraction of teeth 54, 52, 51, 61, 62 and 64. An OPG from the initial consultation and bitewing radiographs taken at the time of GA are shown in Figures 7 and 8. Of note are the large carious radiolucencies in teeth 54 and 64 with intimate association of the underlying developing teeth 14 and 24 with the trifurcation area of

Figure 10. *Intra-oral photographs taken at the time of the second general anaesthesia when the patient was six years and ten-months-of-age demonstrating severe caries in tooth 53 and severe enamel hypoplasia in the prematurely erupted teeth 14 and 24*

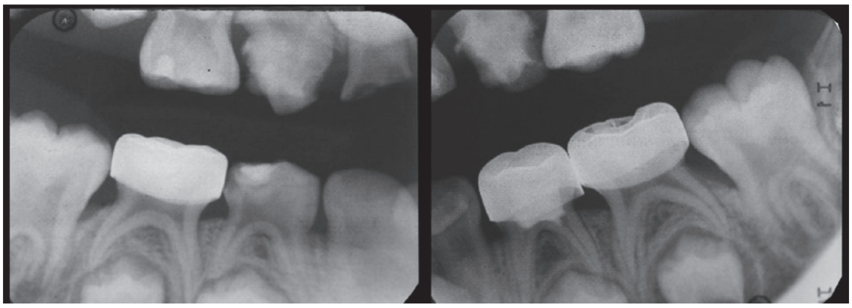

Figure 11. *Bitewing radiographs taken at the time of the second general anaesthesia when the patient was six years and ten-months-of-age.*

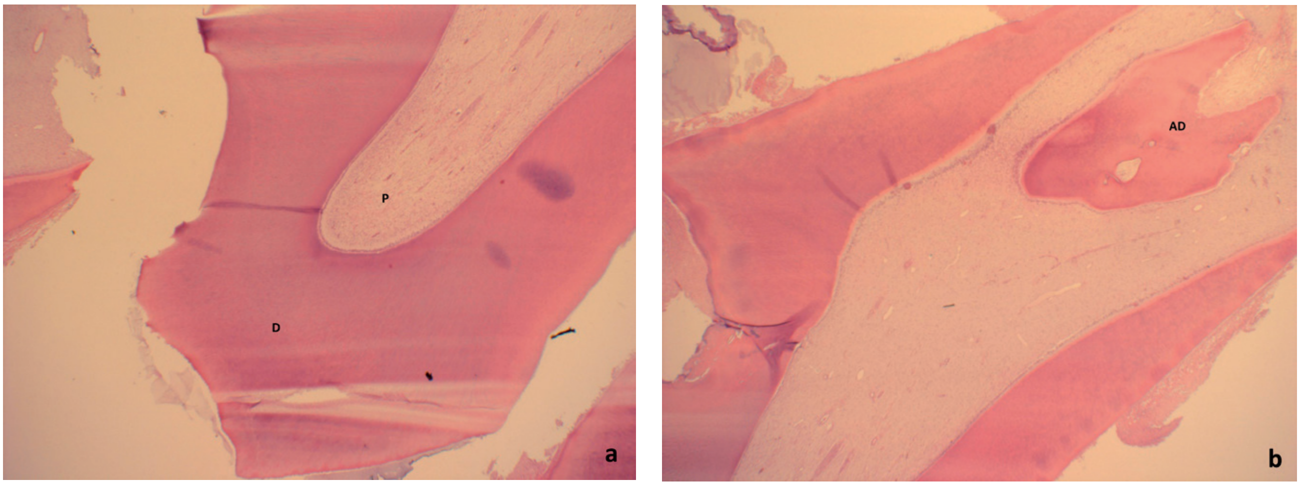

Figure 12. *Photomicrographs of the extracted tooth 14, H & E staining. (a) Relatively normal tubular dentine (D) and vital pulpal tissue (P) apart from localised areas of deficient dentine (arrow) suggestive of overlying severe enamel hypoplasia (b) Atypical atubular dentine (AD) at the bifurcation region, resembling secondary dentine*

Figure 13. *Post-operative review six weeks following general anaesthesia demonstrating fair oral hygiene, sound restorations and good healing of extraction sites. The patient was not cooperative for a full series of photos.*

their primary predecessors. Following dental treatment under GA, the patient had been seen irregularly over the next two years in the Paediatric Dentistry Department for preventive dental treatment. An OPG taken at one of these review appointments (see Figure 9), when the patient was five years and ten-months-of-age, demonstrated enamel hypoplasia and immature root development of the prematurely erupting teeth 14 and 24. At this time, an interim therapeutic restoration was recommended on the partially erupted tooth 24, however, the patient failed to return for the planned treatment until the current presentation. On examination, teeth 14 and 24 were observed to be prematurely erupted with black and brown discolouration and severe enamel hypoplasia exposing softened dentine on multiple surfaces and were tender to percussion with grade one mobility. Additionally, severe carious lesions affecting all surfaces of tooth 53 and cavitated carious lesions extending into dentine in teeth 55, 65, 73, 83 and 84 were noted along with attrition of the 75 preformed metal crown exposing the underlying cement. Owing to the presence of multiple new carious lesions, symptoms and poor patient co-operation, a second GA was deemed necessary to complete dental treatment. Due to the

significant enamel hypoplasia, presence of symptoms, immature root structure, high caries risk and poor compliance, the treatment plan consisted of extraction of teeth 14, 24 along with extraction of the severely carious 53, balancing extraction of tooth 63 and restoration of the other carious teeth and perforated crown on tooth 75.

Under GA at age six years and ten months, a full examination and bitewing radiographs were performed (see Figures 10 and 11). Direct resin-based composite restorations were placed on the labial surfaces of teeth 73 and 83 (Tetric N-Ceram A1, Ivoclar Vivadent, Schaan, Liechtenstein) and preformed metal crowns (3M™ESPE™, NSW, Australia) were fitted on teeth 55, 65, 75 and 84 after caries removal and conventional crown preparation. Local analgesia (4.0 mL lignocaine 2% 1:80 000 adrenaline) was administered via submucosal infiltrations followed by simple elevation and forceps extraction of teeth 14, 53, 63 and 24. Teeth 14 and 24 were subsequently preserved in 10% buffered formalin and sent for histopathological examination. After decalcification of tooth 14 and staining in haematoxylin and eosin (H&E), microscopy revealed a vital premolar with incompletely formed roots and focal abnormal dentine in the bifurcation area (see Figure 12).

The four-week post-operative review revealed good oral hygiene, sound restorations and good healing of extraction sockets (see Figure 12). Ongoing reviews and preventive treatment are planned on a three-monthly basis with monitoring of growth and development until the patient is at an appropriate age and level of compliance to treatment plan for the missing teeth 14 and 24.

Discussion

Enamel hypoplasia

Enamel hypoplasia is a quantitative developmental defect occurring during deposition of the enamel matrix, which is associated with micro- and macroscopic enamel defects[17]. In the present case report, both patients had the most severe type of enamel hypoplasia (type IV) with enamel discolouration and significant loss of normal coronal morphology. These structurally defective teeth are prone to fracture and provide a favourable area for colonization of cariogenic bacteria, particularly in the two patients presented, who had high caries risk and poor oral hygiene. Hypoplastic permanent teeth are reported to be up to seven times more likely to become carious than those teeth without enamel hypoplasia[10,18]. Interestingly, the dentine morphology of the hypoplastic premolars was also noted to be abnormal on histopathological analysis, with focal areas of atypical atubular dentine, resembling secondary dentine. In an investigation of enamel hypoplasia in primary teeth using polarized light and scanning electron microscope, it was reported that the porous enamel, which constitutes a pathway for bacteria and other pulpal irritants, leads to the formation of reparative dentine, which likely accounts for the presence of these focal areas of abnormal dentine[17].

Premature eruption

Premature eruption can be defined as emergence of a tooth into the oral cavity at a time that would be considered early in the context of the child's age, developmental stage, medical history, family, ethnic and genetic background and existing dentition[19-21]. Human dental eruption is regulated by highly complex signalling mechanisms, which is sensitive to various influences on the molecular, cellular and genetic level, and is still not fully understood[19, 22-24]. Altered eruption of permanent teeth is the most common anomaly related to infection in overlying primary teeth [10]. McCormick et al. 1 found the average time for exfoliation and permanent tooth eruption to be a little earlier in abscessed primary teeth than in a control group. If the infected primary tooth is extracted, Posen [25] reported that early eruption of permanent teeth is most likely to occur if this loss occurs within one year of expected eruption time; otherwise, it is more likely that permanent eruption will be delayed.

Severe pulpal infection of primary teeth is known to result in excessive osteolysis of inter-radicular bone and premature eruption of the succedaneous tooth before adequate root length formation[5], which was observed in both patients who had erupted premolars at age six years. One of the proposed mechanisms is that enamel hypoplasia predisposes to a dysfunctional Hertwig epithelial root sheath, which affects cervical constriction and normal root development [26]. Premature eruption of under-developed teeth poses problems such as tooth mobility, discomfort and abnormal migration of the tooth [27]. Unfortunately, there are no known treatments to accelerate the root development of a vital permanent tooth or delay premature eruption. Treatment options for these teeth remain limited, particularly if they are mobile and symptomatic in patients with poor compliance, as in these cases, where extraction was the only predictable treatment option.

Risk factors

Even though there is a high prevalence of primary teeth with peri-radicular inflammation, few permanent successors present with such dramatic structural alterations such as in these cases. A study investigating the effects of experimental pulpal exposures in primary molar teeth of monkeys that were left open for 45 to 212 days discovered spread of inflammatory cells to the follicular tissues of underlying permanent teeth in 21% of exposed molars, but only 10% showed evidence of degenerative changes in the dental epithelium of the developing tooth [11]. These figures are similar to those of Niswander et al.[28], who found 22% of premolars associated with retained primary molar root fragments displayed developmental defects, and Matsumiya [3], who showed that degenerative changes were induced in approximately 20% of developing tooth germs of young dogs following experimental pulp exposure of overlying primary teeth. It would therefore appear likely that, in about one-fifth to one-quarter of infected primary teeth, inflammation might spread into the follicular tissues of the underlying permanent teeth and subsequently induce degenerative changes.

The exact pathogenesis of dysmorphia in the permanent successor related to pathology in the primary tooth is still largely unknown. It is not clear whether the damage occurs by direct bacterial invasion itself or from bacterial chemical mediators or metabolic by-products, or from host-derived proteolytic enzymes such as matrix metalloproteinases [10,14]. There is also the possibility that trauma

during extractions might lead to the developmental defects; however, in case one it is clear radiographically that the follicle of the successor tooth was already abnormal prior to extraction of the primary predecessor. Other studies also support that it is the primary tooth infection that predisposes to the permanent tooth development defects, rather than any treatment performed, such as pulpectomy [29] or extraction[10].

Certain factors have been reported as potentially protective or predisposing with regards to developmental defects in permanent successors and should be considered when determining risk (see Table 2). It has been suggested that when a gingival parulis forms associated with an infected primary tooth, this has a protective effect, which might explain why many severely infected primary molars do not always result in developmental defects of the permanent successor[10]. Observational studies have found that when the onset of peri-radicular tissue inflammation coincides with the precocious stages of the formation of the permanent successor, as was in these cases, the consequences are more significant[10,11]. In both these cases, it appears that the affected premolars were in the Stage B of formation, according to the Demirjian et al.[30] classification. Furthermore, the anatomical position and length of contact between the peri-radicular lesions and the permanent successor also has an influence on the extent of defects[10]. In a histological study of infected primary teeth in monkeys, Andreasen et al.[6] observed that chronic peri-apical inflammation remained confined by a thin fibrous barrier up to a period of at least six weeks, after which it might be unrestricted to result in deleterious changes in the permanent successor. In both these patients, infection in the primary teeth was present for at least two months. So from this limited case series, it can be surmised that severe carious lesions resulting in furcal bony rarefaction adjacent to a primary tooth in a child aged younger than four years, where the permanent successor crown is in the early stages of formation and is intimately positioned in relation to the furcation area of the infected primary tooth, represents a significant risk for severe developmental defects in the permanent successor. Thus, this knowledge should inform treatment planning and consent procedures with families, to encourage timely treatment of such infections.

Prevention

Ultimately, the best prevention of developmental defects in the permanent dentition would be achieved through the optimal prevention and detection of carious lesions in primary teeth and appropriate restorative and pulpal management prior to the development of peri-radicular rarefaction. Once peri-radicular infection is established, the earlier these lesions are detected and treated, in theory, the less the destructive sequelae on the permanent germs. With the renewed interest in non-restorative cavity control[31], these cases serve as an important reminder of some of the significant long-term consequences that can result from untreated dental caries and should be considered during the informed consent process.

These two cases also highlight the poor attendance rate of many high caries risk patients, which likely contributed to the need for repeat dental treatment under GA. More research is required into methods to improve compliance of high caries risk patients with preventive regimes, as literature reveals that children who have dental work completed under GA are more likely to have new carious lesions after the procedure and often require a second procedure, as was in the case of both these patients[32]. These cases further emphasise that dental caries in young, high risk, unco-operative patients needs to be managed aggressively under GA, with full coverage restorations placed where possible and extraction of teeth with questionable long-term prognosis, to avoid the need for repeat GAs. Modification of risk factors for caries should also be a priority.

Restorative options

The aim of treating hypoplastic teeth should be to preserve the pulpal vitality and prevent further tooth structure loss while restoring form, function and aesthetics. In a co-operative patient, with the absence of significant pulpal pathology, enamel hypoplasia can usually be managed by restoring the affected enamel with a bonded restoration, and appropriate monitoring and preventive treatment [33]. Preformed metal crowns are likely to have better longevity in cases of significant enamel loss, but have associated aesthetic consequences[34]. In most cases, especially in young patients and partially erupted teeth, interim therapeutic restorations are necessary until definitive rehabilitation is possible.

Due to limited co-operation of these young patients, and the unreliability of pulp sensibility tests in young children[35], these tests were not performed clinically; however, histopathological examination revealed that the teeth in case two were still vital, and therefore had potential for continued root development. Unfortunately, due to lack of tooth structure and difficultly maintaining restorations on these teeth in a high caries risk, un-compliant patient, the best treatment option was extraction. In a more compliant patient, conservative restoration, temporary splinting and close monitoring for further root development might have been appropriate, as was successfully completed in a case report with seven year follow-up[36]. With the recent evidence-base supporting regenerative endodontic procedures[37], it is possible that successful regeneration of a severely hypoplastic, non-vital and immature tooth, such as in case one,

Table 2. *Factors influencing the development of developmental defects in permanent successors after peri-radicular infection in overlying primary teeth*

Virulence of micro-organisms
Patient immunity
Anatomical position of the permanent tooth bud in relation to the primary tooth roots
Innate protective mechanisms of the dental follicle
Dental age/stage of tooth development
Duration of infection/inflammation
Degree of tissue involvement of the infection
Presence of gingival parulis (draining sinus)

might be viable; however, this was not considered appropriate for this patient due to her inability to cooperate for dental treatment, poor history of compliance with recall visits and the severely compromised tooth structure.

Orthodontic considerations

Caries, infection and premature loss of primary teeth can have significant orthodontic consequences, with a higher likelihood of ectopic eruption of premolars associated with abscessed primary teeth[1]. Space maintenance should be considered in cases of early loss of primary teeth, however, the quality of evidence supporting this practice is low, and unco-operative patients with high caries risk are often contraindicated for the procedure[38, 39]. It is interesting to note that, in case one, there had been minimal loss of space in the lower second premolar regions, even three years after extraction of the lower primary second molars at age three years. This is contrary to the expected belief that severe space loss will occur with early loss of primary second molars, before eruption of the first permanent molars[40, 41]. Unfortunately, for this patient, this means the space in the 35 region cuold persist, necessitating orthodontic space closure or prosthetic replacement in the future. Conversely, in case two, mixed dentition space analysis reveals that it is likely that spontaneous space closure of teeth 14 and 24 spaces will occur with natural eruption of other permanent teeth. In this case, extraction of tooth 63 was performed to balance the extraction of the severely carious tooth 53, in order to prevent a midline shift when the permanent incisors erupt[42]. These two cases draw attention to the need for highly individualised treatment planning when extractions are considered, due to the unique nature of each tooth loss pattern and overall occlusion.

Conclusion

Developmental defects in the permanent dentition can have devastating implications for aesthetics, function, comfort, occlusion and overall patient wellbeing. Prevention of caries and infection in the primary dentition is paramount, as is its timely management should it occur, in order to optimise the prognosis of the permanent successors. Further investigations are required to determine which types of patients, teeth and infections are most likely to cause severe enamel hypoplasia or premature eruption in the permanent successors and the pathogenic mechanisms, to better develop preventive and management options. From this limited case series, it can be recommended that if a child aged younger than four years presents with a severe carious lesion in a primary molar with evidence of pulpal infection in proximity of a permanent successor in the early stages of crown development, the family should be warned of the possibility of severe developmental defects and associated consequences in the permanent dentition.

Why is this paper important to paediatric dentists?

- This paper provides a review of the complications that can occur in permanent teeth subsequent to caries and infection in primary teeth and protective and predisposing factors
- This paper serves as a reminder that families should be warned of the devastating complications that can be inflicted on permanent teeth subsequent to pathology in primary teeth in young children
- This paper highlights the need for further behavioural research regarding compliance of high caries risk patients, particularly those requiring treatment under general anaesthesia

REFERENCES

1. McCormick, J. and D.J. Filostrat, Injury to the teeth of succession by abscess of the temporary teeth. J Dent Child, 1967. 34(6): p. 501-4.
2. Turner, J.G., Two Cases of Hypoplasia of Enamel. Proc R Soc Med, 1912. 5(Odontol Sect): p. 73-6.
3. Matsumiya, S., Experimental pathological study on the effect of treatment of infected root canals in the deciduous tooth on growth of the permanent tooth germ. Int Dent J, 1968. 18(3): p. 546-59.
4. Valderhaug, J., Periapical inflammation in primary teeth and its effect on the permanent successors. International Journal of Oral Surgery, 1974. 3(4): p. 171-182 DOI: 10.1016/S0300-9785(74)80022-0.
5. Kalra, N., Sequelae of neglected pulpal infections of deciduous molars. Endodontology, 1994. 6: p. 19-23.
6. Andreasen, J.O. and I. Riis, Influence of pulp necrosis and periapical inflammation of primary teeth on their permanent successors: Combined macroscopic and histologic study in monkeys. International Journal of Oral Surgery, 1978. 7(3): p. 178-187 DOI: 10.1016/S0300-9785(78)80022-2.
7. Mass, E., I. Kaplan, and A. Hirshberg, A clinical and histopathological study of radicular cysts associated with primary molars. Journal of Oral Pathology & Medicine, 1995. 24(10): p. 458-461 DOI: 10.1111/j.1600-0714.1995.tb01134.x.
8. Kalra, N., K. Sushma, and G.K. Mahapatra, Changes in developing succedaneous teeth as a consequence of infected deciduous molars. Journal of the Indian Society of Pedodontics and Preventive Dentistry, 2000. 18(3): p. 90-94.
9. McDonnell, S.T., H. Liversidge, and M. Kinirons, Temporary arrest of root development in a premolar of a child with hypodontia and extensive caries. International Journal of Paediatric Dentistry, 2004. 14(6): p. 455-460 DOI: 10.1111/j.1365-263X.2004.00582.x.
10. Cordeiro, M.M. and M.J. Rocha, The effects of periradicular inflamation and infection on a primary tooth and permanent successor. J Clin Pediatr Dent, 2005. 29(3): p. 193-200.
11. Winter, G.B. and I.R.H. Kramer, Changes in periodontal membrane, bone and permanent teeth following experimental pulpal injury in deciduous molar teeth of monkeys (Macaca irus). Archives of Oral Biology, 1972. 17(12): p. 1771,IN17,1779-1778,IN18,1779 DOI: 10.1016/0003-9969(72)90241-5.
12. Aguilo, L. and J.L. Gandia, Dentigerous cyst of mandibular second premolar in a five-year-old girl, related to a non-vital primary molar removed one year earlier: a case report. J Clin Pediatr Dent, 1998. 22(2): p. 155-8.
13. Yawaka, Y., et al., Delayed eruption of premolars with periodontitis of primary predecessors and a cystic lesion: a case report. Int J Paediatr Dent, 2002. 12(1): p. 53-60.
14. Gupta, S.S., et al., Role of inflammation in developmental odontogenic pathosis. Journal of oral and maxillofacial pathology : JOMFP, 2016. 20(1): p. 164-164 DOI: 10.4103/0973-029X.180986.
15. Priya, P.R.G., B.J. John, and E. Indumathi, Turner's hypoplasia and non-vitality: A case report of sequelae in permanent tooth. Contemporary Clinical Dentistry, 2010. 1(4): p. 251-254 DOI: 10.4103/0976-237X.76395.
16. Silberman, S.L., et al., A Simplified Hypoplasia Index. Journal of Public Health Dentistry, 1990. 50(4): p. 282-284 DOI: 10.1111/j.1752-7325.1990.tb02136.x.
17. Sabel, N., et al., Polarized light and scanning electron microscopic investigation of enamel hypoplasia in primary teeth. Int J Paediatr Dent, 2010. 20(1): p. 31-6 DOI: 10.1111/j.1365-263X.2009.01006.x.
18. Vahid Golpaygani, M., et al., An Evaluation of the Rate of Dental Caries among Hypoplastic and Normal Teeth: A Case Control Study. Research Journal of Biological Sciences, 2013. 4(4): p. 537-541.
19. Kjaer, I., Mechanism of human tooth eruption: review article including a new theory for future studies on the eruption process. Scientifica, 2014: p. 1-14 DOI: https://dx.doi.org/10.1155/2014/341905.
20. Glowacki, J. and K. Christoph, Gender differences in the growing, abnormal, and aging jaw. Dental Clinics of North America, 2013. 57(2): p. 263-80 DOI: https://dx.doi.org/10.1016/j.cden.2013.01.005.
21. Almonaitiene, R., I. Balciuniene, and J. Tutkuviene, Factors influencing permanent teeth eruption. Part one–general factors. Stomatologija, 2010. 12(3): p. 67-72.
22. Cobourne, M.T. and P.T. Sharpe, Diseases of the tooth: the genetic and molecular basis of inherited anomalies affecting the dentition. Wiley Interdisciplinary Reviews, 2013. 2(2): p. 183-212 DOI: 10.1002/wdev.66.
23. Frazier-Bowers, S.A., C.P. Puranik, and M.C. Mahaney, The Etiology of Eruption Disorders—Further Evidence of a Genetic Paradigm. Seminars in Orthodontics, 2010. 16(3): p. 180-185 DOI: 10.1053/j.sodo.2010.05.003.

References continued on opposite page.

24. Rhoads, S.G., H.M. Hendricks, and S.A. Frazier-Bowers, Establishing the diagnostic criteria for eruption disorders based on genetic and clinical data. American Journal of Orthodontics & Dentofacial Orthopedics, 2013. 144(2): p. 194-202 DOI: 10.1016/j.ajodo.2013.03.015.
25. Posen, A.L., The effect of premature loss of deciduous molars on premolar eruption. The Angle orthodontist, 1965. 35: p. 249-252.
26. Kumakami-Sakano, M., et al., Regulatory mechanisms of Hertwigs epithelial root sheath formation and anomaly correlated with root length. Exp Cell Res, 2014. 325(2): p. 78-82 DOI: 10.1016/j.yexcr.2014.02.005.
27. Fanning, E.A., Effect Of Extraction Of Deciduous Molars On The Formation And Eruption Of Their Successors. The Angle Orthodontist, 1962. 32(1): p. 44-53 DOI: 10.1043/0003-3219(1962)032<0044:Eoeodm>2.0.Co;2.
28. Niswander, J.D. and C. Sujaku, Relationship of enamel defects of permanent teeth to retention of deciduous tooth fragments. Journal of dental research, 1962. 41: p. 808-814.
29. Coll, J.A. and R. Sadrian, Predicting pulpectomy success and its relationship to exfoliation and succedaneous dentition. Pediatr Dent, 1996. 18(1): p. 57-63.
30. Demirjian, A., H. Goldstein, and J.M. Tanner, A new system of dental age assessment. Hum Biol, 1973. 45(2): p. 211-27.
31. Gruythuysen, R.J., Non-restorative cavity treatment: should this be the treatment of choice? reflections of a teacher in paediatric dentistry. Dental Update, 2019. 46(3): p. 220-228 DOI: 10.12968/denu.2019.46.3.220.
32. Berry, E.J., Effectiveness of a Preventive Recall Strategy for Children After Dental Rehabilitation with General Anesthesia. Pediatric Dentistry, 2017. 39(7): p. 450-454.
33. Mink, J.R. and S.A. McEvoy, Acid etch and enamel bond composite restoration of permanent anterior teeth affected by enamel hypoplasia. J Am Dent Assoc, 1977. 94(2): p. 305-7 DOI: 10.14219/jada.archive.1977.0277.
34. Seale, N.S. and R. Randall, The use of stainless steel crowns: a systematic literature review. Pediatr Dent, 2015. 37(2): p. 145-60.
35. Kayalvizhi, G., Reliability of pulp vitality testing in children: a review. Vol. 2. 2008. 259-266.
36. Atabek, D., et al., Maturogenesis of an Early Erupted Immature Permanent Tooth: A Case Report With 7-Year Follow-Up. The Journal of Clinical Pediatric Dentistry, 2015. 39(3): p. 262-267 DOI: 10.17796/1053-4628-39.3.262.
37. Sachdeva, G.S., et al., Regenerative endodontic treatment of an immature tooth with a necrotic pulp and apical periodontitis using platelet-rich plasma (PRP) and mineral trioxide aggregate (MTA): a case report. Int Endod J, 2015. 48(9): p. 902-10 DOI: 10.1111/iej.12407.
38. Laing, E., et al., Space maintenance. International Journal of Paediatric Dentistry, 2009. 19(3): p. 155-162 DOI: 10.1111/j.1365-263X.2008.00951.x.
39. Law, C.S., Management of premature primary tooth loss in the child patient. Journal of the California Dental Association, 2013. 41(8): p. 612-8.
40. British Orthodontic Society. Managing the developing occlusion. 2010 [cited 2019 30 May]; Available from: https://www.bos.org.uk/Portals/0/Public/docs/Making%20a%20Referral/Managing-the-Developing-Occlusion-Updated-Apr10.pdf.
41. Finucane, D., Rationale for restoration of carious primary teeth: a review. European Archives of Paediatric Dentistry, 2012. 13(6): p. 281-92 DOI: 10.1007/BF03320828.
42. Rock, W.P., Extraction of primary teeth - balance and compensation. International Journal of Paediatric Dentistry, 2002. 12(2): p. 151-153 DOI: 10.1046/j.1365-263X.2002.332_2.x.

Australia and New Zealand Society of Paediatric Dentistry
www.anzspd.org.au

President	Dr Sue Taji fed.president@anzspd.org.au
Vice President	Dr Soni Stephen fed.vicepresident@anzspd.org.au
Secretary	Dr Carmel Lloyd fed.secretary@anzspd.org.au
Treasurer	Dr Rod Jennings fed.treasurer@anzspd.org.au
Immediate Past President	Dr Tim Johnston timjohnston@westnet.com.au

Branch Executives

Branch	President	Secretary	Fed Councillor	Treasurer
NZ	Dr Mike Brosnan nz.president@anzspd.org.au	Dr Craig Waterhouse nz.secretary@anzspd.org.au	Dr Heather Anderson russell.heather@xtra.co.nz	Dr Craig Waterhouse nz.treasurer@anzspd.org.au
NSW	Dr Prashanth Dhanpal nsw.president@anzspd.org.au	Dr Jason Michael anzspd.nsw@gmail.com	Dr Soni Stephen sonistephen71t@gmail.com	Dr Maansi Juneja nsw.treasurer@anzspd.org.au
QLD	Dr Sonali Mistry drmistry@outlook.com	Dr Gregory Ooi go.65@optusnet.com.au	Dr Sue Taji drsuetaji@qdg4kids.com.au	Dr Gregory Ooi go.65@optusnet.com.au
SA	Dr Alice Howarth howarth.alice@gmail.com	Dr Nina Yuson sa.secretary@anzspd.org.au	Dr Michael Malandris manjmichael@gmail.com	Dr Gabrielle Allen gabrielle.j.allen@gmail.com
VIC	Dr Giselle D'Mello giselle.d'mello@rch.org.au	Dr Kelly Oliver secretary.anzspdvb@gmail.com	Dr Daniel Andreassen-Cocker dcockertoo@icloud.com	Dr Debra Elsby vic.treasurer@anzspd.org.au
WA	Dr Mark Foster wa.president@anzspd.org.au	Dr Joy Huang wa.secretary@anzspd.org.au	Dr Carmel Lloyd wa.fedcouncillor@anzspd.org.au	Dr Greg Celine wa.treasurer@anzspd.org.au

Editor Synopses	Steven Kazoullis steven@kazoullis.com
Correspondence	Steven Kazoullis PO Box 6253, Fairfield Gardens, QLD 4103
Artwork, printing and distribution	Colgate

Synopses is proudly sponsored by
Colgate Oral Care
Level 14, 345 George Street, Sydney NSW 2000 AUSTRALIA

Mailing List
The mailing list for the distribution of Synopses is maintained by Dr John Winters on behalf of the Federal Secretary/Manager of ANZSPD. It is compiled from information supplied by the Branch Secretaries. If there are errors in your mailing details, please contact Dr John Winters or your Branch Secretary.
Please do not contact Colgate for address correction.

Submissions
All text for inclusion in Synopses must be submitted to the editor by email.
Address email to steven@kazoullis.com
Please include your contact details with all submissions.

UPCOMING EVENTS

28-30 November 2019
4th Meeting of IADR, Asia Pacific Region
Brisbane Convention and Exhibition Centre
iadr-apr2019.com

5 December 2019
Abstracts due for
ANZSPD-Colgate Research Award
bit.ly/2CCBGhT

6-7 March 2020
ANZSPD 20th Biennial Congress
Hotel Grand Chancellor, Hobart
anzspd2020.com.au

21-24 May 2020
2020 AAPD 73rd Annual Session
Nashville, Tennessee
annual.aapd.org

12-14 June 2020
Paediatric Dentistry Association of Asia
12th Biennial Congress
Seoul, South Korea
pdaa2020.org

17-20 June 2020
IADT 21st World Congress on Dental Traumatology
Lisbon, Portugal
iadt-dentaltrauma.org

1-4 July 2020
EAPD 15th Congress of the European Academy of Paediatric Dentistry
Hamburg, Germany
eapd2020.eu

7-12 June 2021
28th IAPD Maastricht Congress
Maastricht, The Netherlands
iapd2021.org